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GROWTH AND DEVELOPMENT OF TEETH AND JAWS (A) MANDIBLE (B) MAXILLA*

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THE mandible, at birth, is more or less a thin shell of growing bone, the body of which seems almost entirely composed of the crypts of the developing teeth. In addition to these many hollow spaces which enclose the tooth follicles the bulk of bone is further reduced to a state of porosity by the abundance of vessels present. The course of these vessels indicates the direction of growth. During this early stage of development the distribution of bone in the mandible is such as to support the body in all directions, protecting the developing teeth, affording attachment for operating muscles, and preventing distortion during active functional period of nursing and swallowing. The significance of the distribution of the spongy and forming compact bone will be pointed out later. The design of the mandible at birth is one example of many of the intimate relations between form and function to be found in the body.

The maxilla articulates with two bones of the cranium and seven bones of the face; thus strongly supported, it remains in a fixed position, the mandible being the movable part of the facial bones. In the mechanics of nursing, the gum pads do not come into contact with each other but remain separated vertically during active periods of nursing, approaching one another no nearer than the thickness of the compressed nipple. The muscular action of the mandible is principally in the forward and backward direction of sucking, rather than in the up and down movement of chewing. It is, therefore, a distinct advantage for the ramus to be at an obtuse angle of about one hundred and seventy-five degrees to the body of the mandible (Fig. 1) and for the mandibular fossa to be more or less flat, corresponding to or harmonizing with the forward and backward function. Even in a state of rest the gum pads are separated by the infant's tongue.

The vault or roof of the mouth is comparatively flat.

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As no teeth have erupted in either jaw, the tongue is not confined as it is in the complete dentition; so, in a state of rest, it extends over the ridges of the gum pads, maintaining its place between the upper and lower jaws. This is an opportune time for the development of the deciduous teeth which are lying in their crypts just under the occlusal pads, and additionally protected by the soft overlying tongue.

As the child grows older and the deciduous incisors begin to erupt, the process is apparently accompanied by an itching, uneasy sensation which causes the child to bite upon some object to gain relief. This vertical biting movement stimulates and accelerates the eruption of the incisor teeth and encourages the beginning of a change in the angle of the jaw, which gradually approaches that of a right angle, and the contour of the mandibular fossa takes form.

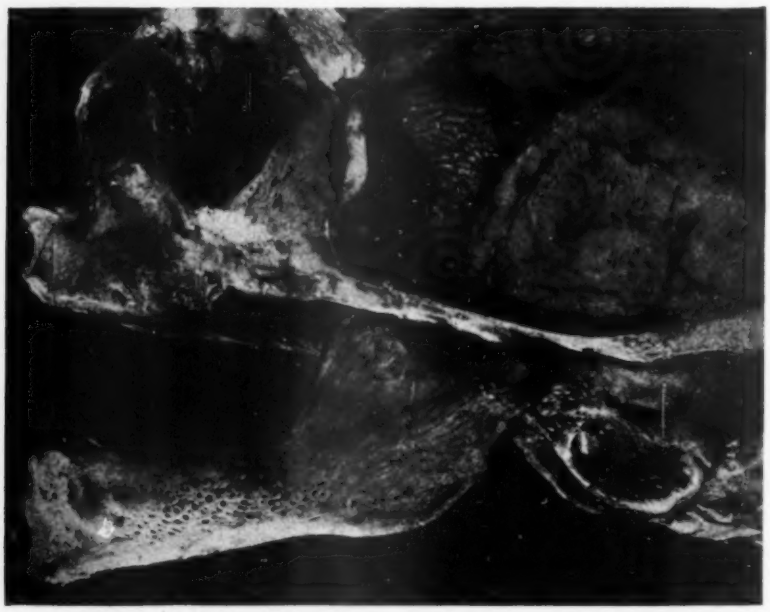

Fig. 1.—Mandible at birth, in position of function.

By the time the child requires food which necessitates breaking up or chewing, the incisors and first deciduous molars have come into occlusion; thus the gum pads posterior to these teeth are kept apart, preventing injury to the follicles of the posterior teeth lying just below the mucous covering (Fig. 2). The increased stimulation of this biting force associated with the continuous growth of the alveolar process of the erupted deciduous teeth, raises the occlusal plane, thereby further separating the jaws and increasing the height of the face. The existing disproportion between the upper and the lower half of the face is in this manner eliminated in favor of the lower half.

In the course of normal development and tooth formation there arises, within the first weeks of embryonic life, a thickening of the epithelium at the free margin of the jaws, which is called the dental lamina, and which runs backward, maintaining at first its location. Corresponding to each deciduous tooth this dental lamina proliferates and forms five buds in each half of either jaw. Later, on the lingual side of these buds, as the primary dental lamina breaks

up, the so-called secondary laminae grow, the deeper parts of which develop into buds of the corresponding permanent teeth.

The bud for each permanent molar develops directly from the primary dental lamina distal to the follicles of the second deciduous molars.

At birth the crypts of the permanent anterior teeth begin to form.

At about the age of *five months* the first anatomic-osteologic indication of the crypts of the first premolar may be observed, which bud, as mentioned, is given off lingually to the primary dental lamina of the first deciduous molar. It rests in a tiny cavity on the inner surface of the lingual plate of the mandible near the top border, just opposite and above the center of the lingual surface of the first deciduous molar.

In a mandible at *six months* of age, a horizontal section is made about midway from the lower to the upper border of the body. The crypts of the permanent incisor teeth and canines are already formed, lying directly behind or lingual to corresponding deciduous teeth, giving the appearance of a double row of teeth (Fig. 3).

Fig. 2.—Incisors in occlusion, and first deciduous molars in the state of eruption.

In a mandible *twelve months* of age, we find the crypts of the second premolar developing in the same manner as that of the first premolar at the age of five months, on the inner surface of the lingual plate, but deeper and more sharply defined (Fig. 4). At this time a part of the crown of the second deciduous molar is partially covered by bone; the same is true in the case of the first deciduous molar at six months of age. This overlying bone becomes resorbed as eruption continues; simultaneously the lingual and buccal plates become thicker, leaving the tooth follicle lying on top of the lingual plate of the mandible (Fig. 5).

The follicle of the developing premolar, however, is more or less protected from external injury by the rounding lingual surface and lingual inclination of the mandibular deciduous molar. Its location at the age of 24 months may be seen in Fig. 6*A* and *B*.

The deciduous molar teeth do not begin active occlusal movement until the calcification of the bifurcation is started, or is in process of completion. At this time the formation of the spongy bone between the roots of the deciduous molars begins. This spongy bone is in intimate contact with the mandibular canal (Fig. 7). The bone between the bifurcation of the roots of the molars grows upward with the crowns of the teeth (Fig. 8). As these erupt, the

Fig. 3.—Mandible 6 months of age: the entire body is taken up by the crypts of the developing teeth.

Fig. 4.—Specimen 12 months of age; shallow excavation at the upper border of the lingual plate receiving the germ of the second premolar, *A:* section of buccal plate removed, showing crypt of deciduous second molar.

spongy bone between their roots increases in an upward direction and covers the buccal side of the bony crypts of the premolars. (Fig. 6*A* and *B*.)

Up to this time there has been no alveolar process. This process develops progressively with the eruption of the deciduous molars. The crypts of the premolars are left behind, being embedded in the true lingual wall of the

Fig. 5.—Specimen 12 months of age: sockets of the second deciduous molar, showing the spongy bone between its roots and the developing alveolar process, both enveloping by their union the tooth follicle, *A*.

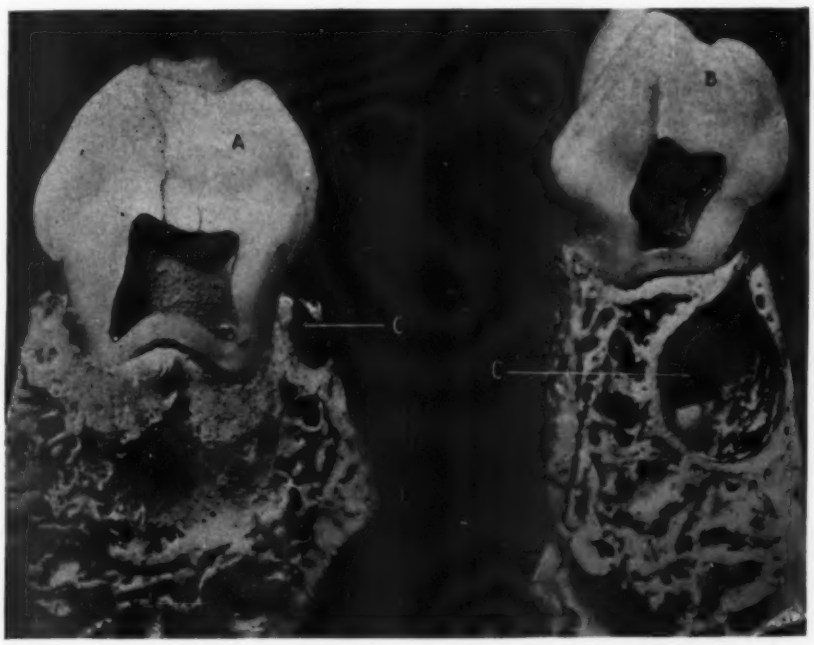

Fig. 6.—*A*, specimen 24 months of age, showing crypt of developing second premolar, *C*. *B*, crypt of first premolar more advanced than that of the second, *A*.

Fig. 7.—The bifurcation of the deciduous molar being complete, the formation of the trabecular arrangement of bone has begun, being in intimate contact with the mandibular canal; *A*, this bone configuration is characteristic for the active state of eruption.

Fig. 8.—Cancellous bone grows up with the crown of the tooth. See text for explanation.

mandible and not in the alveolar process. This becomes increasingly evident since the growth of the alveolar process is at a more rapid rate than that of the lingual or buccal plates of the mandible. The changing relationship of the alveolar crest to the crypts of the developing teeth gives one the impression of the crypts descending into the body of the mandible.

The small tooth crypt lying in the lingual wall of the mandible may be seen in actual cross sections, though not in an x-ray of an unsectioned jaw, due to the interception of the rays by the deciduous molar.

In the case of the absence of the follicle of the premolar (Fig. 9) the spicules are so arranged as to support the tooth normally in the walls of the mandible.

Fig. 9.—Absence of follicle of the premolar; see text for explanation.

At about the age of *24 months* the shallow open pocket or seat of the tooth crypt seemingly begins to descend, leaving behind a small opening, the gubernaculum; the lower portion of the crypt takes on a bulbous shape which gives the appearance of a hanging, elongated, drop of oil. (Fig. 10).

The tooth follicle now grows rapidly. At the age of about *30 months* it has moved downward along the lingual wall of the mandible, and inward between the roots of the temporary molar. (Fig. 11.)

At the age of *2½ and 3 years,* respectively, the crypts of the first and second premolars, which are in contact with the lingual plate of the mandible, have enlarged to such an extent as to be well within the body of the mandible, between the roots of their respective predecessors.

At about *6 years* of age the size of the crypts of the developing premolars, and their downward and buccal growth, seems to be completed (Fig. 12). The gubernaculum remains.

At this time of development, when the crypt has completed its descent, the actual eruption of the premolar begins. There seems to be some uncertainty as to whether the crypt of this developing tooth actually moves downward and inward into the body of the mandible, or whether the sides of the mandible, growing upward, leave the base of the crypt in its original location. However that may be, the eruption of the tooth and the development of the alveolar process take place at a faster rate than does the growth of the lingual and buccal plates of the body of the mandible. Hence the tooth is able to erupt, forcing the crown through the gum tissue well above the upper border of the plates of the mandible.

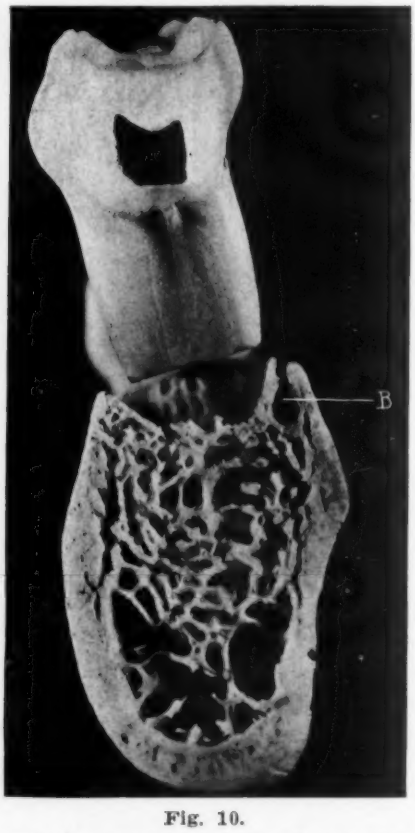

Fig. 10.

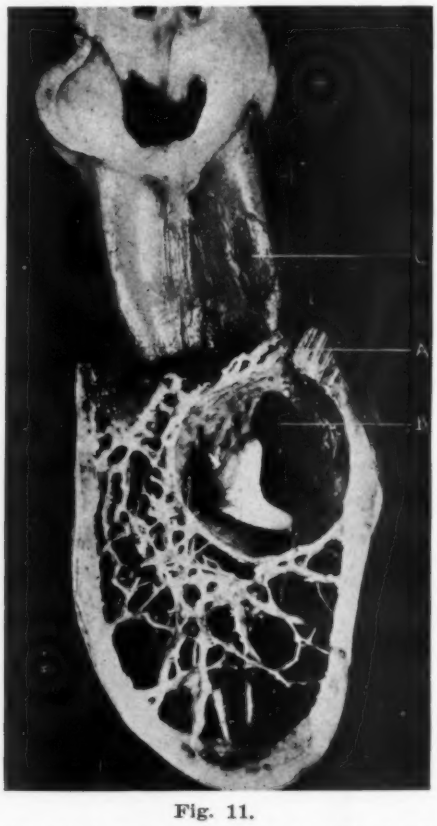

Fig. 11.

Fig. 10.—Specimen 24 months of age; the typical pear shape of the bud is quite significant.

Fig. 11.—Specimen 28 months of age. Note the gubernaculum opening into the crypt of the developing second premolar, *A*, and the open space into the tooth crypt caused by the encroachment of the root of the second deciduous molar; *B*, the portion of the developing root of this molar, which intruded into the crypt of the developing follicle, is resorbed, as shown in *C*.

Cross sections of developing mandibles demonstrate the apparent downward growth of the tooth crypts, the beginning of calcification of the permanent teeth, and their eruption. Fig. 13 is a specimen two years of age; sections were made at *A*, *B*, *C*, *D*, *E*, and *F*, corresponding to the different teeth.

In section *A*, the crown of the permanent lateral incisor is almost calcified, and now the tooth is ready to begin its eruption directly to the lingual of the deciduous tooth.

B is a section through the middle of the deciduous and permanent canines. Here again the crypt has descended as far as the lower border of the mandible; the crown being calcified, the tooth is ready to start its movement opposite to that of the crypt; the ascent—the eruption—starts, following the gubernaculum to the lingual of the deciduous canine.

In *C*, the crypt of the first premolar has begun its first movement—its descent—while the cusps begin to calcify. The calcified portions have followed the downward and inward descent of the tooth crypt until its lowest point has been reached. During the course of this descent the crown has become completely calcified, and now begins the opposite movement—the ascent.

Fig. 12.—Specimen 6 years old. Crypt of the developing premolar completed.

Fig. 13.—Specimen two years of age; cross sections through the different teeth: *A*, lateral incisor; *B*, canine; *C*, first deciduous molar; *D*, second deciduous molar; *E*, first permanent molar; *F*, second permanent molar; *G*, crypt of second molar.

In *D*, the small crypt of the second premolar has formed to the lingual of the second deciduous molar and has begun its downward and inward descent to take its position between the roots of its predecessor. This is a very critical time in the development of the premolar, because of its exposed position to inflammation or mechanical injury which might occur in this region.

In *E*, the section is made in the mid-portion of the first permanent molar. As the bud of this tooth is given off to the distal of the dental lamina, the crypt is begun on the upper surface of the mandible midway between the buccal and lingual walls. The completely calcified crown and the degree of the descent may be seen in the cut. This crypt also descends into the body of the mandible, and when it has reached its lowest point the crown has calcified and will, like the other teeth, begin its ascent.

In *F*, the section is made through the developing crypt of the second permanent molar. This crypt is given off, as was that of the first permanent molar, on top of the body of the mandible and to the distal of the first permanent molar. This beginning crypt may be seen at point *G*.

Fig. 14.—Specimen 7 years of age: cross sections through the different teeth; *A*, central incisor; *B*, lateral incisor; *C*, canine; *D*, first premolar; *E*, second premolar; *F*, mesial root of first permanent molar; *G*, bifurcation of first permanent molar; *H*, second permanent molar; *I*, distal part of the crypt of the second molar; *K*, crypt of the third molar.

Similar sections are made of a specimen about *7 years* of age (Fig. 14). The permanent central and lateral incisors and the first permanent molars have erupted. In *A* and *B* will be seen the extent to which the roots of the central and lateral incisors have ascended. In *C*, the canine tooth is now almost ready to ascend in the process of eruption; the crown is fully formed, and the crypt has descended and taken its place in the lower portion of the mandible, encroaching somewhat upon its lower border. The crowns of the first and second premolars are fully formed and calcified and are ready for eruption.

A section through the mesial root of the first permanent molar and another in the area between the two roots is shown in *F* and *G*. Note the wide apex of the yet incompletely formed root and the spicules underneath in this area; compare and pay special attention to the difference in the structure and arrange-

ment of the bony spicules beneath this root (Fig. 14*F*), and in the bifurcation of the two roots (Fig. 14*G*). The latter is destined to support the weight of the occlusal forces as long as the roots are not fully formed and able to offer resistance. The spicules in the bifurcation are much more numerous and the individual spicules are thicker. The functional structure of this arrangement offers the maximum resilient support. The bony spicules beneath the root are scarce and thin and do not show such a functional architecture. It reminds one of the characteristic different architectural structure of the supporting bone in functioning and nonfunctioning teeth.[1]

In *H*, the cross section, in this instance, is made through the center of the second permanent molar. Note the relation of this bifurcation to the mandibular canal which lies contiguous to the floor of the crypt.

In *I*, the cross section is made through the distal of the second permanent molar crypt, showing the opening, or small depression, which is to be the seat of the developing third molar (*K*).

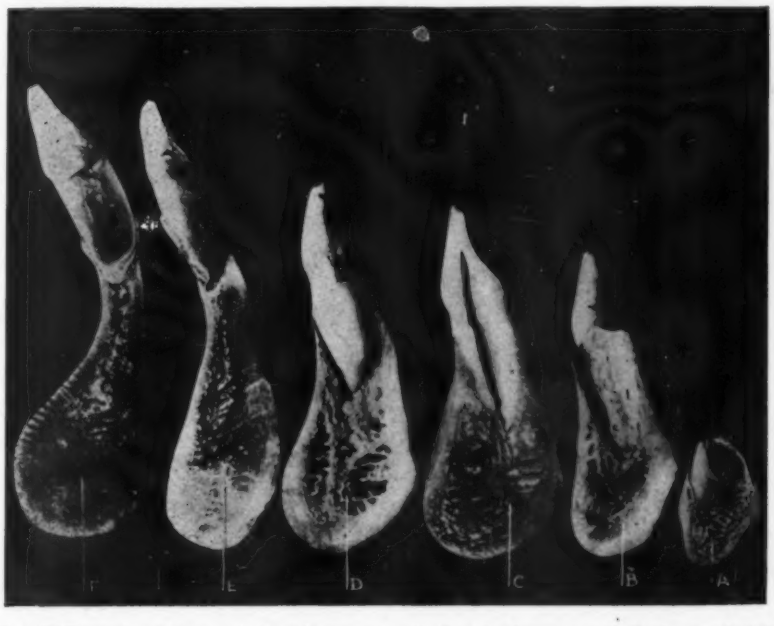

Fig. 15.—Different stages of development of the incisors and their pertaining alveolar process: *A*, two months; *B*, seven years; *C*, nine years; *D*, eleven years; *E*, sixteen years; *F*, twenty-five years.

In each instance, from the central incisor to and including the third molar, the buds have been given off and the crypts have begun their development on the upper border, and not down in the body, of the mandible; the crypts of the incisors, canines, and premolars develop on the *upper border of the lingual plate of the mandible,* while the permanent molars also originating on the upper border of the mandible are situated *between the buccal and lingual plates.*

Six mandibles were selected and dissected to illustrate the growth of the alveolar process and the upward and forward ascent of the incisor teeth from the former position of their crowns at the lower border of the mandible (Fig. 13 and Fig. 14). These sections were made in the region of the midline (Fig. 15); *A* represents the state of development at 2 months, *B* at 7 years, *C* at 9 years, *D* at 11 years, *E* at 16 years, and *F* at 25 years.

For purposes of comparison, another series of mandibles was selected and cut longitudinally.

So we see in Fig. 16 a specimen estimated to be one year of age; the buccal plate of bone was removed so as to show the development of the teeth and the distribution of the cancellous bone within the two plates of the mandible. The anterior descending border of the ramus meets the body of the mandible just above the center of the second deciduous molar; the lower border of the mandible has a rather dense cortical plate, which is necessary for support during function. The angle of the mandible, however, does not have such a support. The abundance of cancellous bone present indicates this is the region of rapid growth. The different directions of the spicules and vessels, seemingly coordinated, indicate the different directions of growth and development of the mandible.

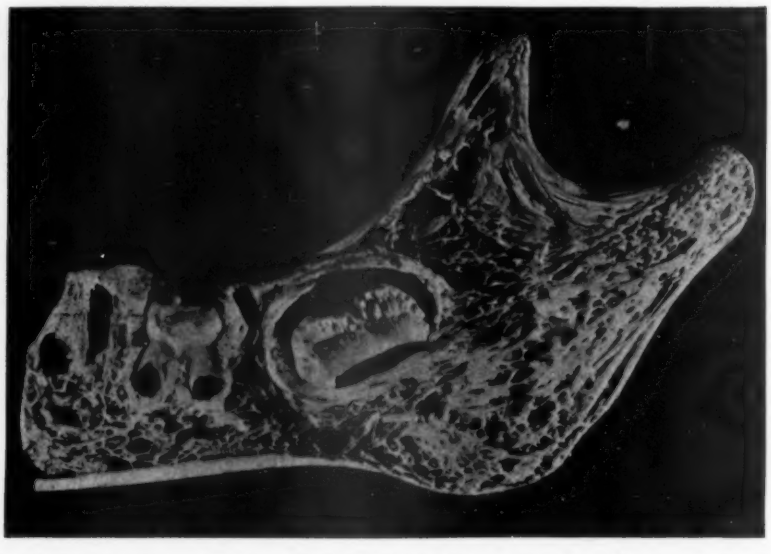

Fig. 16.—Specimen 1 year of age; longitudinal section; outer plate removed. See text for explanation.

According to my opinion, the bone of the jaws in the state of active development is very much more porous than that of the adult or of old age. The compact plate of bone on the lower border of the mandible indicates the rapidity of development has diminished; further growth takes place principally in the process upward and at the angle of the ramus.

Fig. 17 represents the mandible of a child *6 years* of age, with the crypts and crowns of the developing teeth. At this age the anterior descending border of the ramus meets the body of the mandible to the distal of the first permanent molar, indicating the extent of the lengthening of the body of the mandible at six years of age.

In comparing Fig. 17 with Fig. 16, at *1 year* of age, where the anterior border of the ramus meets the body just above the center of the second deciduous molar, we see that the mandible has lengthened the width of nearly one and one-half molar teeth.

Fig. 18 represents the mandible of a child approximately *13 years* of age. The permanent teeth have all erupted now, except the third molar; the apices

of the second molar and the second premolar are not yet fully formed. The amount and distribution of the cancellous bone correspond to normal conditions for this age. The lower border of the mandible is supported by quite dense cortical bone which does not yet extend completely to the angle of the mandible, indicating that growth is still continuing in this latter region. In other words, the vertical development of the face at the age of thirteen is progressing more rapidly, together with the forward growth of the jaw. The anterior border of the ramus now meets the body of the mandible distal to the second permanent molar.

Fig. 17.—Specimen 6 years of age. See text for explanation.

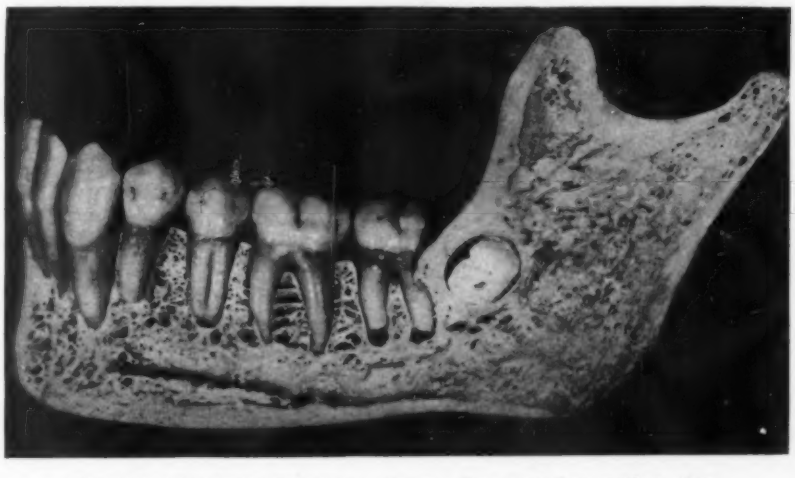

Fig. 18.—Specimen 13 years of age. See text for explanation.

In comparison with Fig. 17 (6 years), the mandible has further increased in length by another width of a molar, while in comparison with Fig. 16 (1 year) the difference amounts now to the width of two and one-half molars.

In Fig. 19 the mandible of an adult is reproduced. All teeth, including the third molar, have erupted. The anterior border of the ramus at this state of development meets the body of the mandible to the distal of the third molar. By

comparing this figure with Figs. 16, 17, and 18, we obtain a clear idea of the extent to which the body of the mandible has grown in mesiodistal direction during the period from one to twenty years.

The lower border of the mandible is composed of a thick layer of cortical bone which extends to and around the angle, up the distal border of the ramus to the neck of the condyle. This presence of compact bone indicates, from the anatomic standpoint, the cessation of growth though not of form. The vessels in the mandibular canal of this specimen are mummified, and small branches may be seen leading off to the apices of the molar teeth. Note that the branches of these vessels lie in bony channels similar to the main branches of the mandibular nerves and blood vessels.

As in Fig. 15, one may also note here the extent to which the complete set of teeth has erupted, increasing gradually the distance of the apices from the lower mandibular border. It is also a noteworthy phenomenon that the distance of the mandibular canal from the lower border is gradually increasing during the periods of growth. (Compare Figs. 17, 18, and 19.)

Fig. 19.—Specimen of an adult. See text for explanation.

Comparing Fig. 14 with Fig. 19, the extent to which the crown of the canine has traveled in the course of development and eruption may be noted. Actually, each tooth travels occlusally at an even faster rate than the alveolar process develops. If this were not so, the crown of the tooth could never emerge beyond its upper border. The alveolar process develops with the eruption and is dependent upon the presence of the teeth which it supports.

To Be Continued

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407 Professional Building

ORTHODONTIC RESULTS MANY YEARS AFTER TREATMENT

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WHEN Dr. Noyes invited me to present a case report, he had several things in mind. The three most important among them were: (1) That I should show a case or cases "many years after the appliances had been removed," (2) That the case or cases should be in any of the classes of malocclusion "with the exception of Class II Division 1," and (3) "That the time permitted is forty-five minutes" which, as indicated on the program, has been cut down to thirty minutes. With demands and restrictions of this sort, it is necessary to be economical with ideas, time, and material, all of which are rather abundant when orthodontic experiences cover a period of more than thirty years.

To meet such contingencies and at the same time contribute some useful information, I shall limit my presentation to the general concept of *aims in orthodontic treatment and the means of attaining them.* The first and all-important aim in orthodontia, as all Angle graduates understood it before orthodon*tics* took the place of orthodon*tia,* was the establishment of normal occlusion of the teeth in dentitions with malocclusion. The original idea was that, by putting the teeth in normal occlusion, "nature" is given a chance to be of help in the normal development of the other structures surrounding the teeth; namely, the alveolar processes, the jaw bones, and the rest of the face. This, of course, was the inference. What would actually happen was really not known then. But after the lapse of years, it was gradually learned just what does happen. With the needed proof so abundantly available, it is now easy enough to demonstrate some actual facts. It should, however, be remembered that the presentation of results many years after they have been accomplished is likely to meet with some complexities. Changes in technique and changes in point of view are potent factors in molding opinions. Those who participated in these changes are mindful of what the original techniques were, what they are now, and how they came about. But those who happen to tumble into them now are not only less informed but are also bound to be less critical and less exacting. As it appears to me, the aim of orthodontic treatment, at present, is not as strictly observed as it was thirty years ago. For example, the case shown in Fig. 1 should at this time present no great difficulties in the attainment of a very satisfactory result. The great strides made in the improvement of orthodontic appliances and technique should warrant that. The treatment of this case as indicated by the date was started in 1931. The best and latest appliances were, no doubt, used, but the aim apparently was not quite clear. After seven and a half years of treatment the result obtained, as shown in Fig. 2, is not what would be considered very satisfactory.

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Read before the Annual Meeting of the American Association of Orthodontists, Chicago, May 15, 1940.

The malocclusion is not changed from what it was. It is still a case of Class II Division 2. Besides this, some of the teeth, maxillary right lateral incisor and mandibular left first molar, are in rather precarious positions. I am showing this case as a sample of a very large number of cases treated by orthodontists throughout this country. In my estimation the result indicates lack of orthodontic aim and orthodontic outlook. To emphasize this fact, I shall show another sample of a like case, but satisfactorily treated. In Fig. 3 are thus shown the casts of a case of malocclusion belonging to the same class.

Fig. 1.—Casts of case of malocclusion, Class II Division 2 subdivision, before treatment.

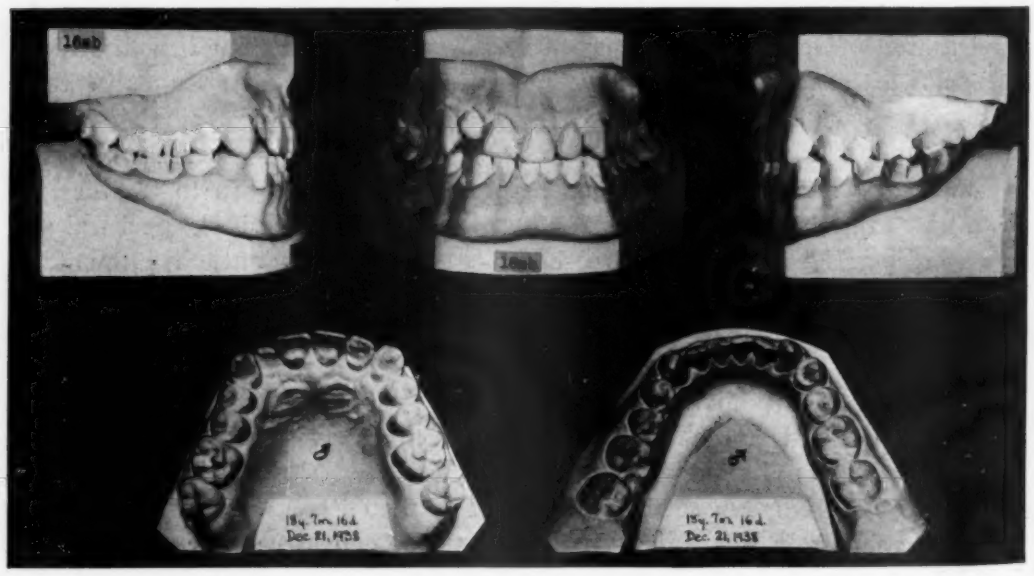

Fig. 2.—Casts of same case as in Fig. 1, showing result after aimless treatment for seven and one-half years.

The difficulties entailed in treatment were very great. But the result obtained, as shown in Fig. 4, is very gratifying despite the accidental loss of a mandibular incisor. This case is used just to emphasize the fact that cases of this sort need not end in disaster if the orthodontic objective is clear and the desire to reach it is strong enough. At the present time, one should certainly not be handicapped by lack of devices, improper technique, or insufficient skill.

Fig. 3.—Casts of case of malocclusion similar to that shown in Fig. 1.

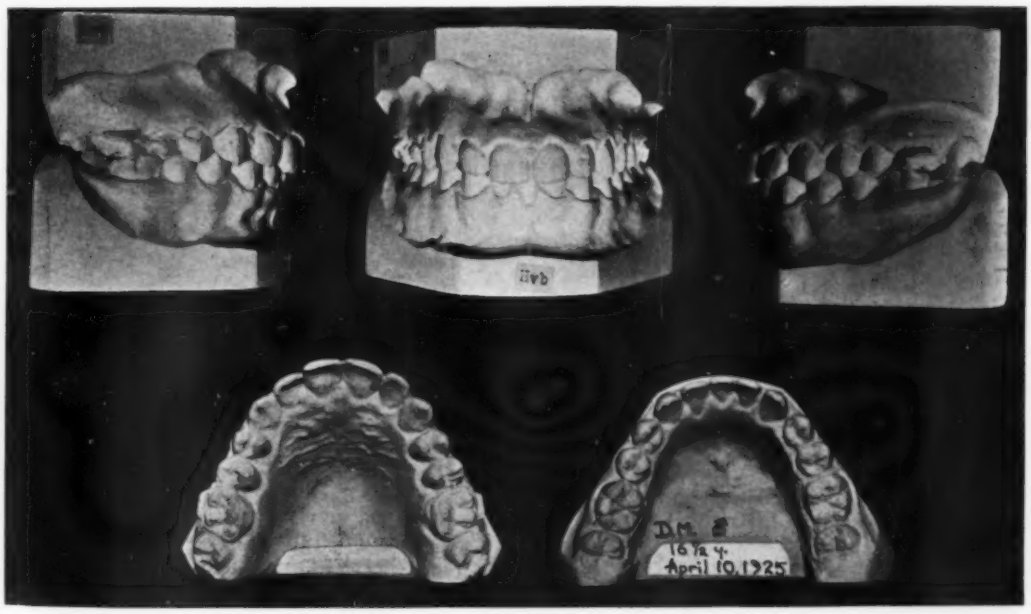

Fig. 4.—Casts of same case as in Fig. 3, six and one-half years later, showing result of successful treatment.

In contrast to this sort of case, I shall present several other cases treated at the very beginning of my orthodontic experience. In Fig. 5 are the dental casts of a girl, 7½ years old, with a Class I case of malocclusion. As shown by the date on the cast, treatment was started in December, 1913. In those days, the choice of appliances was not great, but the aim in treatment was very definite. The appliances used were still the old D band, E arch, and

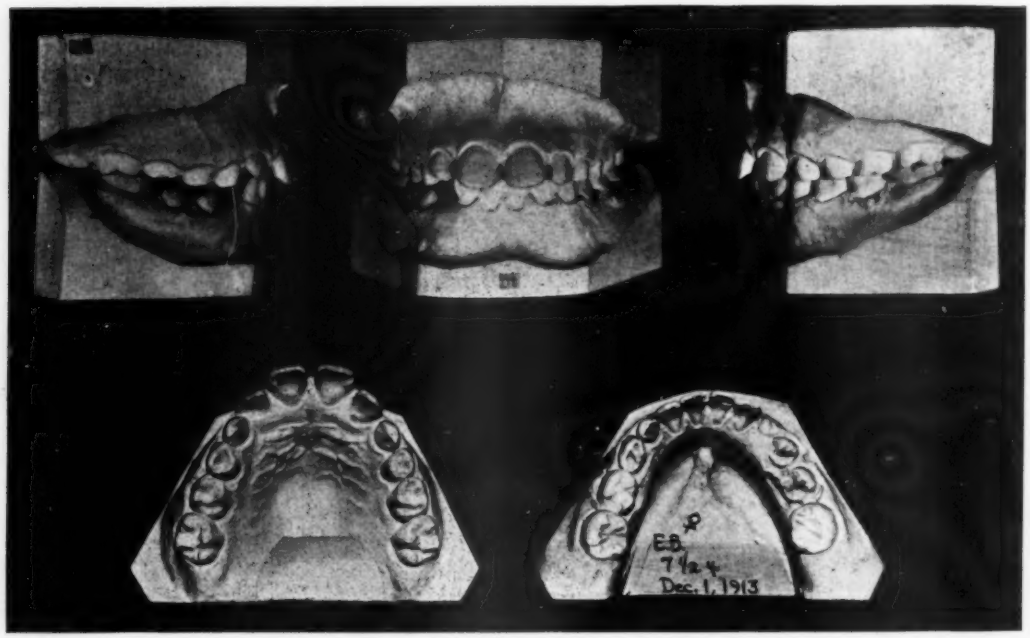

Fig. 5.—Casts of case of malocclusion, Class I, before treatment.

Fig. 6.—Casts of same case as in Fig. 5, three and one-half years later, showing result of successful treatment.

brass ligatures. After three and a half years of treatment, as shown by the casts in Fig. 6, normal occlusion had been established. The lower retainer, as shown in this figure, is still on. It had to be kept on for at least six months. The question then was: "What will happen next?" The answer usually given was: "Leave the rest to nature." How long? Well, it was anybody's guess. In December, 1939, twenty-two and a half years later, the answer was furnished. In Fig. 7, the casts made from impressions at that time bear the proof. "Nature" obviously did well. But the background for doing her share was, no doubt, furnished by the result obtained at first.

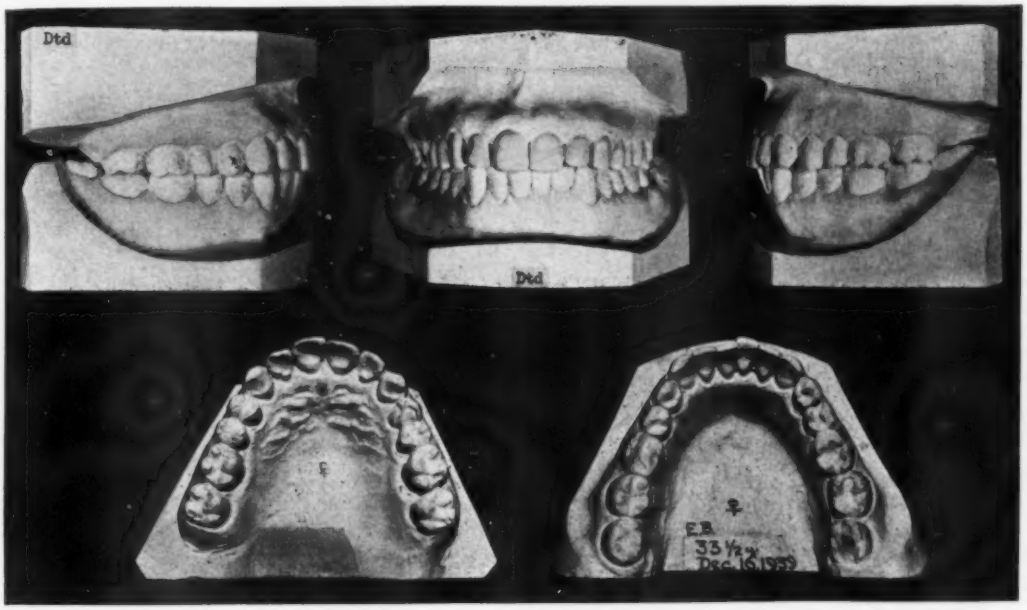

Fig. 7.—Casts of same case as in Figs. 5 and 6, twenty-two and one-half years later, showing effect of time and function.

In Fig. 8 are shown the casts of another case started even earlier, August, 1909. The appliances used were again the "German silver" D bands and E arch with brass ligatures. After eight months of treatment, as shown in Fig. 9, I got into difficulties. Much to my chagrin, the maxillary front teeth were flaring out more than I really liked. Of course, in such cases especially, we used to depend on "nature" for much of the needed help. But I was somewhat uneasy about it in this case. The lateral incisors were too much in horizontal position to be of much comfort. The "pin and tube" appliances were not yet available at that time. I did not cherish putting too great a burden on "nature" either. So I thought that by putting some sort of stop at the incisal edges of those teeth, I might be able to change the situation. I did that by means of a wire fastened to the arch and pulling the tooth at the neck with a ligature. In this way, the root of the tooth was actually carried forward more than the crown. Of course, soldering to the nickel-silver arch was not possible. The "finger" had to be soldered to a tube and the tube slipped onto the arch and soft soldered in place. Anyway, it worked. It took three years to do it, but in August, 1913, the normal occlusion aimed for was established, as shown in Fig. 10, and all the incisor teeth were, what I considered

then and still do now, in proper vertical position. I then felt that leaving the matter to "nature" at this stage would not entail such hardships as before. In January, 1935, twenty-one years later, the opportunity offered itself for me to see the outcome. The casts shown in Fig. 11 furnish the evidence of what happened. As shown by these casts, the occlusal relationship remained normal. But due to conditions outside the realm of orthodontic influence some of the teeth were lost. There is a slight relapse in both the alignment

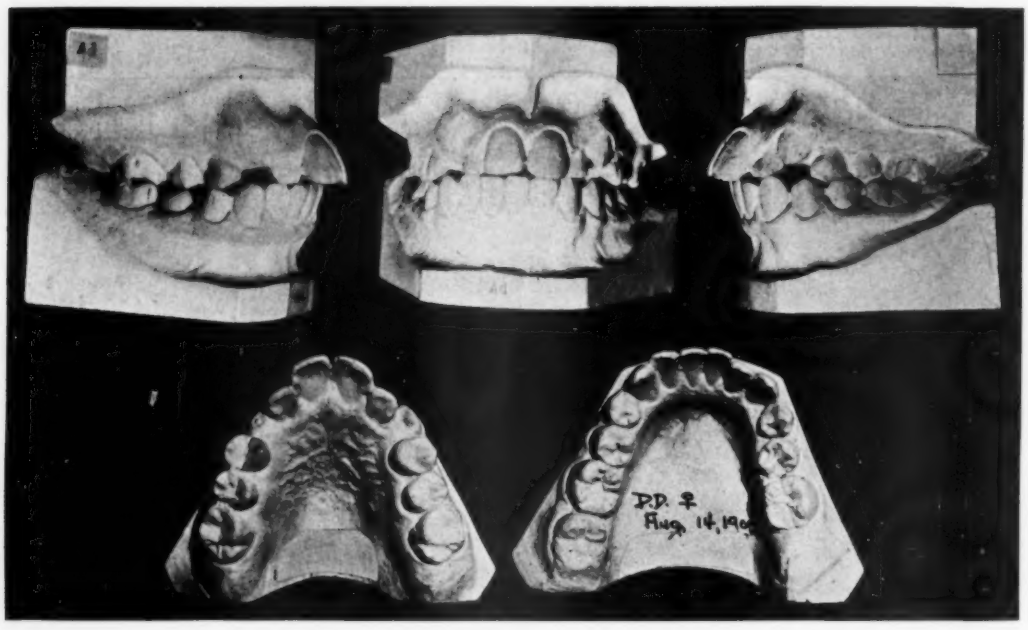

Fig. 8.—Casts of case of malocclusion, Class I, before treatment.

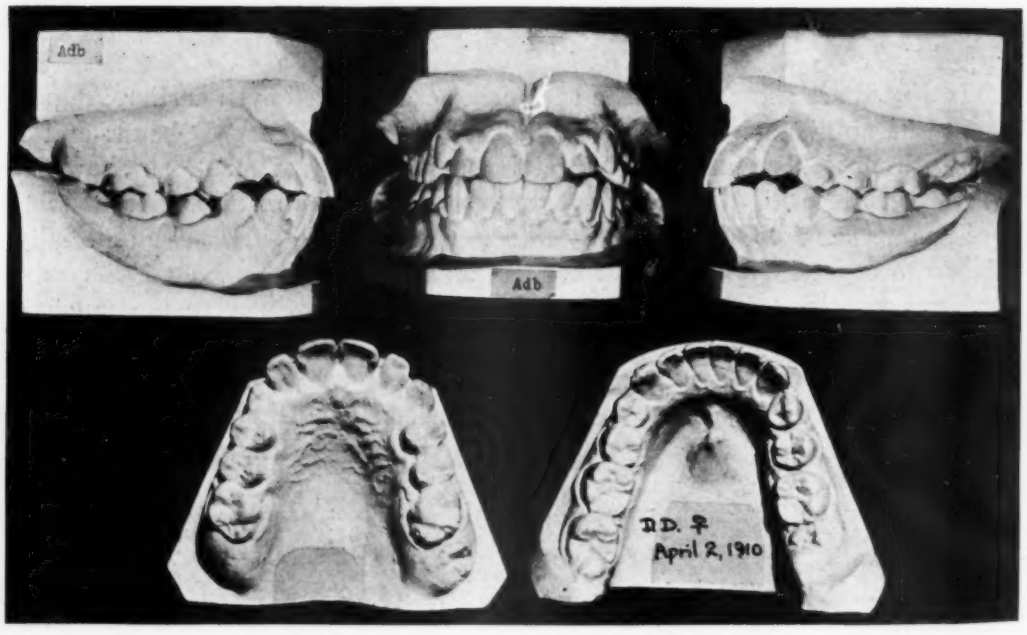

Fig. 9.—Casts of same case as in Fig. 8, eight months later, showing "flaring out" of maxillary front teeth brought about by treatment.

and vertical position of the maxillary incisor teeth. Whatever else "nature" might have done, she was not very generous in this case.

In showing the case in Fig. 12, I shall violate the restrictions imposed. But since the gain is of some advantage, I shall take the chance even at the

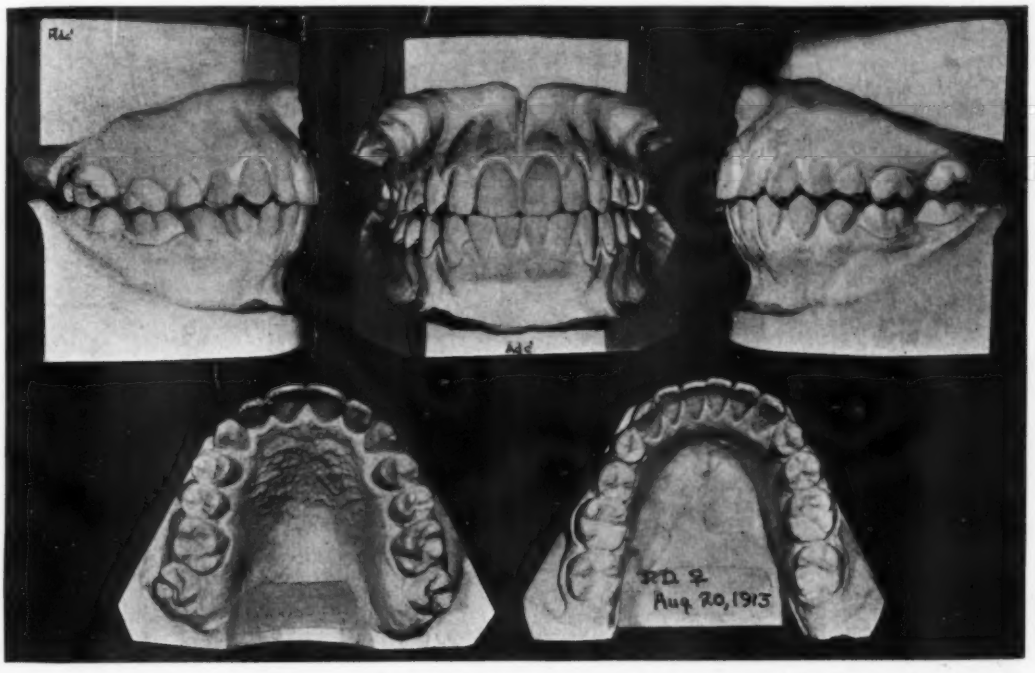

Fig. 10.—Casts of same case as in Figs. 8 and 9, three years later, showing result of successful treatment in occlusion and vertical position of maxillary incisor teeth.

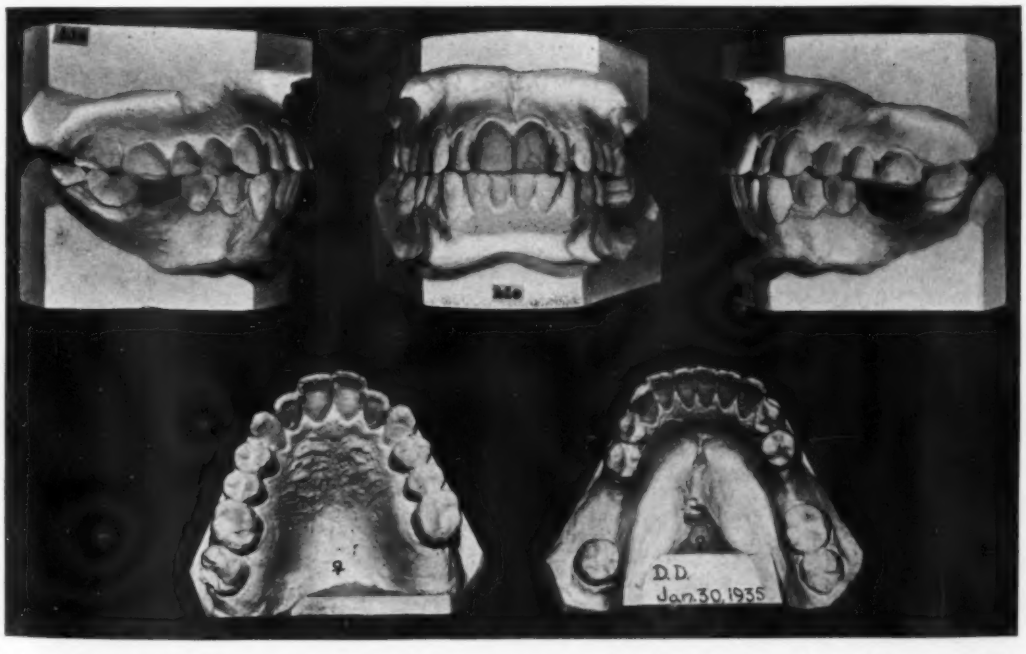

Fig. 11.—Casts of same case as in Figs. 8, 9, and 10, twenty-one years later, showing effect of time and unfavorable conditions.

risk of punishment. As shown by the casts in this figure, this was, at that time, the most extreme case of Class II Division 1 that had come under my care. Treatment was started in August, 1911. The appliances used were the same as in the other cases. But intermaxillary elastics were added. Four years later, the occlusion, as shown in Fig. 13, was normal. This is one of those cases where we used to keep our fingers crossed in hope and fear, praying that it might not have to be done over and over. My anxiety was finally

Fig. 12.—Casts of case of malocclusion, extreme Class II Division 1, before treatment.

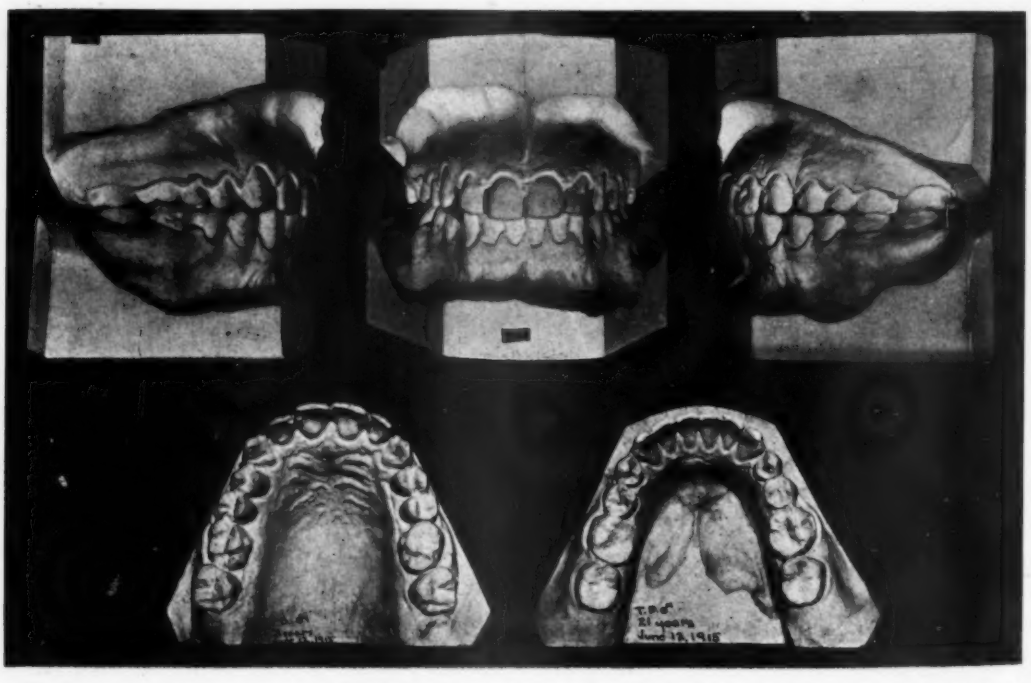

Fig. 13.—Casts of same case as in Fig. 12, four years later, showing result of successful treatment.

relieved when in October, 1934, nineteen and a half years later, I had the chance of taking impressions and making the casts shown in Fig. 14. I know quite well that there are always some among us who have critical suggestions. But there is little need for them now, because the patient is now forty years of age and very grateful for what was done for him.

The last case I shall now show is the first case I had in practice. Fig. 15 illustrates just what it looked like. If anyone imagines that I approached the task of treating it with much pleasure, it is a mistake. I was very enthusiastic, very eager, and extremely ambitious at the time, but terribly scared. The treatment of this case, one might say, an extreme Class III, was a test for the best of us. I plunged headlong into it in 1909. About a year later, as shown in Fig. 16, I was in distress. The trouble: overtreatment on the left side and flaring of the maxillary incisor teeth. At this time the "pin and tube" appliance had just made its appearance. I tried it. Result: devitalization of maxillary right central incisor. I forthwith removed the "pin and tube" appliance and resorted to the same means as in the cases above. After four

Fig. 14.—Casts of same case as in Figs. 12 and 13, nineteen and one-half years later, showing effect of time and "nature."

years of further effort, the result obtained, and a very gratifying one it was, is shown in Fig. 17. This was in January, 1914. What "nature" had in store for me had to be awaited. In February, 1932, eighteen years later, my suspense ended when I took the impressions from which the casts shown in Fig. 18 were made.

The points I should make at this time are:

1. The presentation of these cases is not to be taken as a boast of knowledge, ability, or skill. I really did not know at the time just what I was up against.

Fig. 15.

Fig. 16.

Fig. 17.

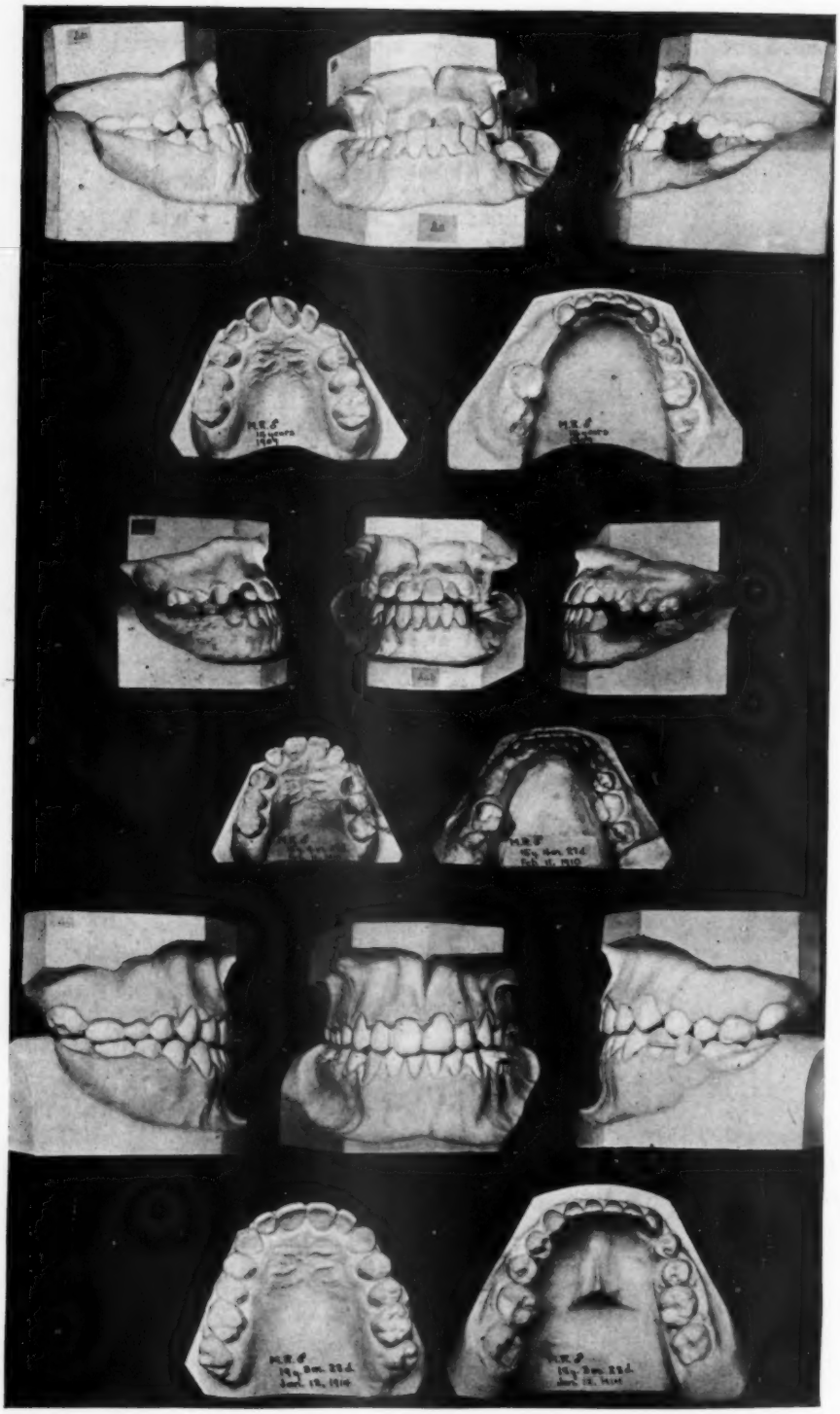

Fig. 15.—Casts of case of malocclusion, Class III. The first case treated by the author.

Fig. 16.—Casts of same case as in Fig. 15, about one year later, showing overtreatment in right first molar region and "flaring out" of maxillary incisor teeth.

Fig. 17.—Casts of same case as in Figs. 15 and 16, showing result of successful treatment.

2. The reason I am presenting these cases is to emphasize the fact, that at the time I undertook the treatment of these and many other cases, I was mainly concerned with (a) the need for a definite goal—normal occlusion—and (b) the determination of attaining it.

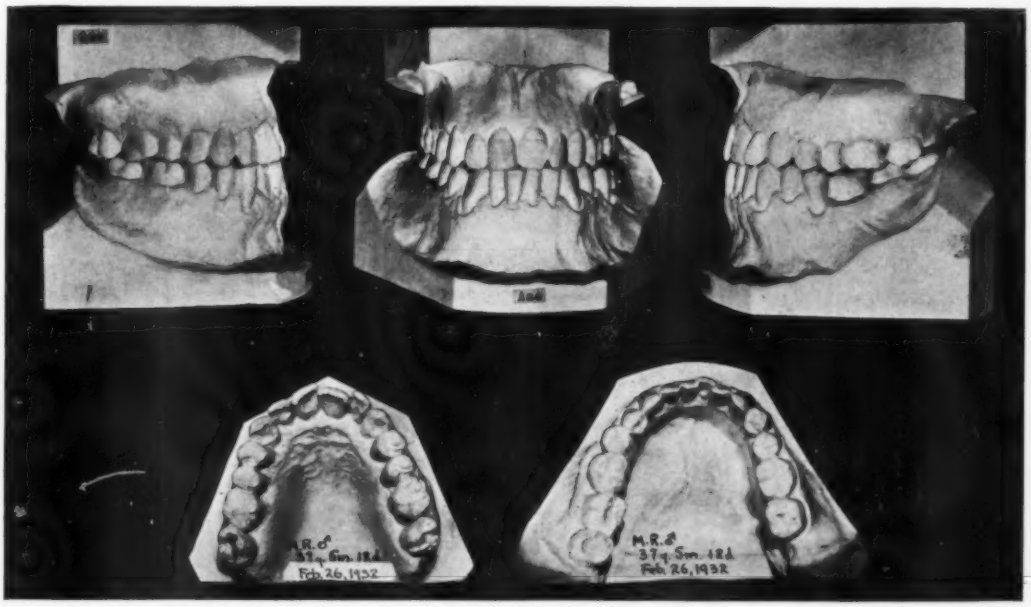

Fig. 18.—Casts of same case as in Figs. 15, 16, and 17, eighteen years later, showing effect of time.

3. The idea of "letting nature do the rest" only appealed to me after I had done all that was needed and that I was able to do. In these respects, the changes in the world of today have not changed my orthodontic outlook of yesterday. What I am wondering about is that, if the orthodontic outlook of today is as the first case makes it appear, the rapid accumulation of like evidence is not very comforting. Unless our streamlined outlook and air-conditioned aims of the present will change materially, orthodontic progress in the thirty years to come will definitely fail to measure up to that of the thirty years just past.

QUANTITATIVE STUDIES IN THE DEVELOPMENT OF THE RAT MOLAR

II. Alveolar Bone, Cementum, and Eruption*
(From Birth to 500 Days)

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INTRODUCTION AND REVIEW OF LITERATURE

IN THE last two decades several important advances in our knowledge of tooth development have been made. The recognition of continuous eruption (Gottlieb and Orban[5]), the histologic evidence for mesial drift (Stein and Weinmann[10]), the experimental studies of alveolar bone growth and its relation to eruption (Brash[2]), and the quantitative analysis of the growth pattern of enamel and dentine (Schour and Poncher[8]) have enriched our literature and influenced our thinking along the biologic aspects of clinical dentistry.

This study attempts to analyze the growth pattern of alveolar bone and cementum and the eruption pattern of the rat molar, and, at the same time, to test experimentally and quantitatively these and correlated questions in the molar of the living rat. For the purposes of this study the growth pattern of a tooth could be defined as the composite resultant of the growth processes that involve cellular activities that proceed at definite rates and gradients and are distributed along definite paths. The eruption pattern may be defined as the composite resultant of the physiologic processes that involve the chronologic changes in the position of the tooth and that proceed along a definite path and direction according to definite rates and gradients.

The molar of the rat was selected as the test object because its enamel and dentine are limited in growth and because it resembles the human tooth in many other respects. Experimental findings on the rates of apposition of primary and secondary dentine in the rat molar are reported separately (Hoffman and Schour[6]).

Vital staining with alizarin red S serves to demarcate fixed bony points or levels from which subsequent relative positions of the tooth may be measured. This method, therefore, makes it possible indirectly to measure the rate of eruption in histologic sections.

A search of the literature failed to reveal any quantitative measurements on the rate of eruption, or on the rate of apposition of alveolar bone and cementum in the rat molar or in other teeth of limited growth.

*This study was made with the aid of grants from the Carnegie Corporation of New York, and from Mead Johnson and Company. The Wistar Institute of Anatomy kindly made available its collection of rat skulls of the Wistar experimental stock for gross and radiographic analysis.

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METHODS AND MATERIAL

Vital Staining.—This study is based on sixty-three white rats from birth to 500 days of age which were given multiple intraperitoneal injections of a 2 per cent solution of alizarin red S at intervals of two to thirty days (Table I). The dosage was 100 mg. per kilogram for animals up to 200 days of age and 50 to 75 mg. per kilogram for the older animals. These animals were the same as those used in the study of the growth pattern of dentine (Hoffman and Schour[6]), and the same histologic sections were used. The basal diet was uniform.

TABLE I

HISTORY OF 63 WHITE RATS INJECTED WITH ALIZARIN RED S

GROUP NUMBER	TOTAL NUMBER OF ANIMALS	NUMBER OF ANIMALS IN SUBGROUPS	TIME OF INJECTIONS. AGE IN DAYS	AGE IN DAYS WHEN SACRIFICED
I	21	12	1, 8, 22, 36	20-40
		7	9, 23, 30	40
		2	20, 34	40
II	27	10	40, 54	60
		8	62-69	77
		9	79-93	100
III	8	2	100, 114	125
		2	125, 142	148
		2	150, 175	177
		2	184, 202	209
IV	7	2	215, 245, 275, 302	309
		2	303, 329, 370, 402	409
		3	403, 443, 465, 495	502

Gross Studies—Height of Clinical Crown.—Measurements of the clinical crown were made on 116 first molars of dissected jaws of twenty-nine animals ranging in age from 21 to 100 days. Two normal rats were sacrificed every second day from the nineteenth to the fortieth days of life and every week thereafter to the one hundredth day. Similar measurements were also made in the sixty-three animals that were given multiple injections of alizarin red S. The measurements were made, with a spreading caliper graduated in tenths of a millimeter, from the crest of the gingivae to the tips of the mesiobuccal cusps of the maxillary and mandibular first molars. These measurements were taken immediately after death, in order to avoid the possible changes following fixation.

TABLE II

EXTRA-ALVEOLAR LENGTHS OF MANDIBULAR AND MAXILLARY FIRST MOLAR ON THE THIRTY-FIFTH AND THE 1050TH DAY (MADE ON DISSECTED SKULLS)

AGE IN DAYS	UPPER FIRST MOLAR				LOWER FIRST MOLAR			
	SITES MEASURED				SITES MEASURED			
	MESIO-PROXIMAL	DISTO-PROXIMAL	BUCCAL	LINGUAL	MESIO-PROXIMAL	DISTO-PROXIMAL	BUCCAL	LINGUAL
35	1.4 mm.	1.0 mm.	1.0 mm.	1.6 mm.	1.6 mm.	0.6 mm.	0.6 mm.	0.6 mm.
1050	1.4 mm.	1.0 mm.	1.0 mm.	2.1 mm.	1.6 mm.	0.6 mm.	0.6 mm.	1.0 mm.

Measurements of the length of the extra-alveolar portions of the molars (Table II) were made on thirty cleaned normal skulls of the experimental stock of the Wistar Institute.

Roentgenographic Studies.—Roentgenograms of the heads of 137 normal white rats thirteen to 1050 days of age were obtained from the experimental stock of the Wistar Institute and from the control animals of previous experiments. The following measurements were taken: (1) The height of the anatomic crown; (2) the length of the anatomic root; (3) the relative position and the distance of the cemento-enamel junction from the alveolar crest (Table III), the distance between the apices of the lower first molar and the lingual surface of the incisor (Fig. 7).

TABLE III

CHANGES IN SIZE AND POSITION OF THE MANDIBULAR FIRST MOLAR OF THE ALBINO RAT FOR THE FIRST 1,000 DAYS OF LIFE, BASED ON MICROSCOPIC MEASUREMENTS IN ROENTGENOGRAMS OF 137 RATS[1]

AGE IN DAYS	HEIGHT OF ANATOMICAL CROWN IN MM.	WEEKLY DECREASE IN MM.	LENGTH OF ANAT. ROOT IN MM.	WEEKLY INCREASE IN MM.	POSITION OF C.E.J. IN REL. TO ALV. CREST IN MM.	TOTAL LENGTH OVER-ALL IN MM.	WEEKLY INCREASE IN MM.	DISTANCE FROM APEX OF MOLAR TO LINGUAL OF INCISOR	
								MES. ROOT	DIST. ROOT
14	1.28		0.38		−0.88	1.66			
21	1.28		1.13	0.75	−0.33	2.41	0.75		
28	1.25	0.03	1.32	0.19	−0.25	2.57	0.16		
35	1.22[2]	0.03	1.67	0.36	+0.18	2.99	0.42	0.48	0.45
42	1.20	0.02	1.78	0.11	+0.2	2.98	--?		
49	1.17	0.03	1.88	0.10	+0.23	3.05	0.06		
56	1.15	0.02	1.94	0.06	+0.26	3.09	0.04		
63	1.12	0.03	2.03	0.09	+0.27	3.15	0.06	0.60	0.60
70	1.10	0.02	2.09	0.06	+0.28	3.19	0.04		
77	1.08	0.02	2.15	0.06	+0.29	3.23	0.04		
84	1.06	0.02	2.20[3]	0.05	+0.3	3.26	0.03		
91	1.03	0.03	2.27	0.07	+0.31	3.30	0.04		
98	1.01	0.02	2.32	0.05	+0.32	3.33	0.03		
135	0.99	0.004+	2.49	0.03	+0.41	3.48	0.03		
200	0.93	0.005	2.65	0.02	+0.44	3.59	0.01	0.90	0.60
300	0.90	0.003	2.81	0.01	+0.54	3.70	0.01	1.10	1.00
400	0.86	0.002	3.03	0.008	+0.57	3.89	0.01	1.30	1.00
500	0.83	0.002	3.18	0.006	+0.64	4.01	0.004	1.70	1.00
750	0.77	0.001+	3.38	0.005	+0.74	4.15	0.01	1.90	1.25
900	0.74	0.001+	3.53	0.01	+0.77	4.27	0.005	2.50	1.50
1050	0.71	0.001-	3.67	0.002	+0.79	4.39	0.005	2.60	1.60

[1]The measurements were taken at the mesial alveolar crest.
[2]Beginning of secondary dentine in pulpal horns in response to beginning of attrition.
[3]Further elongation of root only by cementum apposition.

In order to make them constant and comparable, the measurements were taken along the vertical axis of the mesial cusps of the mandibular first molar. These measurements were made directly from the x-ray film with a 10X filar micrometer eyepiece and a 2X objective on a dissecting scope. The readings were made in microns and transposed into millimeters. This method was checked with microscopic measurements obtained from ground sections of animals of the same ages and proved to be reliable within a 7 per cent ± error.

Microscopic Studies (Ground Sections) (Fig. 1):

1. Rate of Apposition of Alveolar Bone and Cementum.

Over 1,300 micrometer measurements were made in the first molar between successive alizarin red S effects in the secondary cementum and in the alveolar bone (Tables IV to VII). Each measurement represented the arithmetic mean

of three trials. Trial measurements on the second and third molars indicated results that were similar but somewhat slower in rate and later in time.

The alveolar bone was measured at the crest, the fundus and along the walls (Fig. 1 and Table VII). The measurements on secondary cementum were made along lines parallel to the *cementum canaliculi,* since the latter indicate the direction of cementum growth (Tables V and VI).

TABLE IV

GROWTH OF PRIMARY CEMENTUM IN THE MANDIBULAR FIRST MOLAR OF THE ALBINO RAT FROM 10 TO 500 DAYS

AGE IN DAYS	10-20	20-40	40-70	70-90	90-130	130-175	175-210	210-280	280-365	365-400	400-465	465-500
*Total Width in Microns	1.48	4.06	7.50	9.76	14.24	19.01	22.30	27.55	32.56	34.45	37.35	38.78
Estimated Daily Rate	0.148	0.129	0.115	0.113	0.112	0.106	0.094	0.075	0.059	0.054	0.046	0.041

*Based on 476 measurements in decalcified and ground sections at the level of the gingival third.

TABLE V

SUMMARY OF FINDINGS ON PRIMARY AND SECONDARY CEMENTUM

TYPE OF CEMENTUM		RANGE OF DAILY RATES IN μ	STRUCTURE	INCREMENTAL PATTERN	DURATION OF GROWTH IN DAYS
Primary		0.15-0.041	Acellular No canaliculi Finely fibrillar	Parallel to dentino-cemental junction	Begins on eleventh day. Continues throughout life. Thickness increases from 2 μ at second week to approximately 38 μ at 500 days
Secondary	Early	14.48-8.87	Cellular with embedded P.D.M. fibers and canaliculi	Mirror image of dentine. Increments meet d.c. junction at angle	Thirty-fifth to eightieth
	Late	8.87-0.91		Conical with apex toward fundic bone	Eightieth-throughout life

For the study of the primary cementum, the total width was measured at the gingival third of the root. The rate of growth of primary cementum was so slow that the effects of the alizarin injections fused. The rate was estimated by dividing the total width by the age of the animal in days (Table IV).

2. Rate of Elongation of Root.

Microscopic measurements of the rate of elongation of the root were made (1) between the points along the dentino-cemental junction that are met by the alizarin red S effects of the dentine (Fig. 2) during active dentine apposition and (2) between alizarin red S effects in the secondary cementum that was ap-

posed after dentine had ceased to grow in the apical region of the tooth (Table VI and Fig. 2).

3. Width of Periodontal Membrane.

The width of the periodontal membrane up to 500 days of age was measured from microscopic ground and decalcified sections at representative levels (Table VIII).

4. Rate of Eruption and Direction of Tooth Movement.

The effects of vital staining on the alveolar bone and secondary cementum made it possible to make microscopic measurements of the rate of eruption by two methods (Table IX):

(a) By utilizing the cemento-enamel junction as a fixed point on the tooth and determining the rate at which this point changes in relation to the fixed alizarin red S lines of known periods at the alveolar crests.

(b) By adding the rates of fundic bone apposition and of root elongation (the width of the periodontal membrane at this level remains relatively constant throughout life).

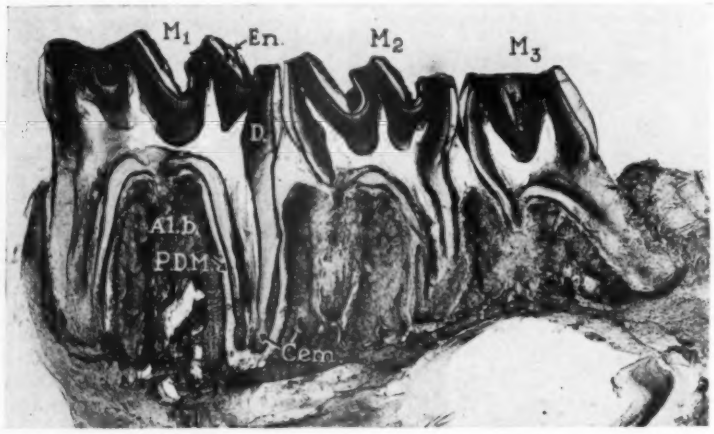

Fig. 1.—Photomicrograph of a mid-sagittal ground section of the molar segment of the mandible of a normal rat 92 days of age. Note the three molars, M_1, M_2, M_3. The section passed through the proximal end of the mandibular incisor. *Al.B*, alveolar bone; *Cem*, secondary cementum; *D*, dentine; *En*, enamel; *P.D.M.*, periodontal membrane. (9.6×.)

FINDINGS

Gross Findings

Height of Clinical Crown.—The vertical height of the clinical crown of the maxillary and mandibular first molars remains relatively constant from the time the opposing teeth interlock (about the thirty-fifth day) up to at least 500 days of age. In the maxillary molars the height of the clinical crown, as measured over the distobuccal cusp along the buccal surface, remains at approximately 1.5 mm. after the thirty-fifth day, with a range of 1.5 to 1.7 mm. There is a gradual increase in height of the clinical crown on the lingual, beginning with the ninetieth to ninety-fifth days.

The clinical height of the mandibular molar along the proximal and buccal surfaces remains constant at about 0.9 mm. The range was between 0.6 and 1.1 mm. and was independent of the various age groups. The lingual surface, however, did increase in height as a result of a slight resorption of the alveolar crest at this area.

TABLE VI

RATE OF ROOT ELONGATION AND RATE OF GROWTH OF ALVEOLAR BONE AND SECONDARY CEMENTUM IN THE MANDIBULAR FIRST MOLAR OF THE ALBINO RAT FROM 14 TO 500 DAYS

AGE IN DAYS	WEEKLY RATE OF INCREASE IN ROOT ELONGATION IN μ	WEEKLY RATE OF FUNDUS BONE GROWTH IN μ	WEEKLY RATE OF ALVEOLAR-CREST BONE GROWTH IN μ (INTERRADICULAR)	WEEKLY RATE OF SECONDARY CEMENTUM GROWTH IN μ
14-21	781.1	54.3	193.1	No growth during
21-35	540.4	53.7	168.4	first 5 weeks
35-49	106.2	51.7	122.8	101.4
49-63	89.7	48.5	94.6	87.3
63-77	58.8	45.5	79.9	61.0
77-91	52.4	41.3	74.5	53.7
91-100	46.6	36.6	70.7	47.2
100-125	38.9	29.8	62.4	39.5
125-200	22.2	20.9	52.3	20.8
200-300	13.4	13.1	41.1	13.2
300-400	10.4	9.8	31.6	10.6
400-500	6.7	6.1	20.9	6.4

These measurements were made for the mesial root of the mandibular first molar. The figures indicated here are obtained from ground sections of sixty-three animals and are averages of the measurements and represent the means since 81.2 per cent of the total number of the measurements approach these means for the site measured.

TABLE VII

SUMMARY OF FINDINGS ON ALVEOLAR BONE GROWTH IN THE MANDIBULAR FIRST MOLAR OF THE ALBINO RAT FROM 14 TO 500 DAYS*

LOCATION			INCREMENTAL PATTERN	RANGE OF DAILY RATE AND GRADIENT OF APPOSITION IN MICRONS	RESORPTION
Alveolar crest	Mesial		Conical and convex	22.4 to resorption during 1st 5 wk.	Adjacent to mesial root
	Bifurcation			Greatest (27.6-3.1)	Mesial corner
	Distal			26.9-2.5	Distal corner
Fundus	Mesial		Flat to concave	7.4-0.6	Distal corner
	Distal			7.7-0.8	Distal corner
Alveolar wall	Mesial wall of mesial root		Parallel with long axis of root	Greatest at mesiofundic corner	Gingival and middle thirds
	Interradicular	Mesial		----------	Middle and apical thirds
		Distal		Continuous 20.7-1.8	None
	Distal wall of distal root			----------	Middle and apical thirds

*Compare these data with those indicated in Fig. 5.

TABLE VIII

SHOWING MEASUREMENTS IN MICRONS* OF WIDTHS OF THE PERIODONTAL MEMBRANE FROM 14 TO 500 DAYS IN THE MANDIBULAR FIRST MOLAR OF THE ALBINO RAT

AGE IN DAYS	CERVICAL THIRD		MIDDLE THIRD		APICES		INTERRADICULAR CREST TO BIFURCATION
	MESIAL	DISTAL	MESIAL	DISTAL	MESIAL	DISTAL	
14	167	170	166	164	132	135	86
40	72	110	140	137	125	128	77
60	65	102	80	118	127	131	78
100	40	96	73	108	127	129	71
200	35	90	66	101	125	129	76
300	31	87	55	84	129	131	81
500	28	81	30	72	128	133	74

*Based on averages of 640 readings.

Length of Extra-Alveolar Portion of Molars.—Measurements on the skulls showed that the extra-alveolar portions of the mandibular first molars remained constant from 35 to 1050 days of age at the distoproximal, the mesial, and the buccal surfaces. Because of resorption there was an increase in extra-alveolar length from the thirty-fifth day to the 1050th day at the lingual surface (Table II and Fig. 7).

Corresponding measurements in the maxillary molars showed the same constancy, although the actual lengths were different from those in the mandibular molars (Table II).

Roentgenographic Findings

Length of Anatomic Crown of the Mandibular First Molar.—The anatomic crown increases until 10 to 14 days of age. It is maximal prior to the abrasion of the enamel, then decreases as a result of attrition in height from 1.27 mm. at 14 days of age to 0.713 at 1050 days. The most rapid attrition occurs during the first 100 days of life, when the crown decreases approximately 0.26 mm. During the next 900 days the crown decreases approximately 0.3 mm., only a little more than the amount of decrease during the first 100 days (Table III and Figs. 7 and 8).

Length of Anatomic Root and Total Length of Tooth.—The length of the root continues to increase from 0.38 mm. at 14 days of age to approximately 3.67 mm. at 1050 days of age. The rate of increase slows down with age. The cementum apposition at the apex which adds to the length of the root continues throughout life and is approximately four times greater than the amount of attrition. The result is a continuous increase in the total length of the tooth (Table III and Fig. 7).

Position of the Cemento-Enamel Junction in Reference to the Alveolar Crest.—The occlusal movement of the cemento-enamel junction is more rapid than that of the alveolar crest. Until the thirty-fifth to fortieth day the cemento-enamel junction lies apically to the alveolar crest. After this period it surpasses the crest and continues to move occlusally (Table III and Fig. 7).

Position of the Apex of the Mandibular Molar.—The apices of the mandibular first molar move occlusally from the lingual of the mandibular incisor with advance in age. The distance between these points for the mesial root is somewhat greater than for the distal root (Table III and Fig. 7). The greatest increases occur after 100 days and appear to result in part from the concomitant increase in axial incline of the molar root in a buccal direction.

Microscopic Findings

Chronology and Rate of Apposition of Primary Cementum.—This cementum begins its formation at about 11 days of age, soon after the apposition of the first layer of radicular dentine. It continues to increase slowly in width from about 2 μ at the second week to about 38 μ at 500 days of age (Table IV). Its growth probably continues throughout life. Its apical extension proceeds with the elongation of the dentinal root till about the fortieth day. Up to this time, for each dentine increment of the root a corresponding portion of primary cementum is apposed at the new root surface that has arisen as a result of the new dentine layer. Thus the dentino-cemental junction comes into being by the

concomitant formation of dentine and cementum, the dentine preceding the cementum slightly.

The primary cementum is acellular. Its matrix contains fine fibrillae. Its increments are parallel with the dentino-cemental junction and the long axis of the root (Fig. 5). The effect of the vital staining with alizarin red S consists of a single continuous line that extends from the cemento-enamel junction to the apical end of the dentinal root. The rate of apposition of primary cementum is so slow that individual staining effects in the case of multiple injections are fused. It follows a gradient which decelerates with age.

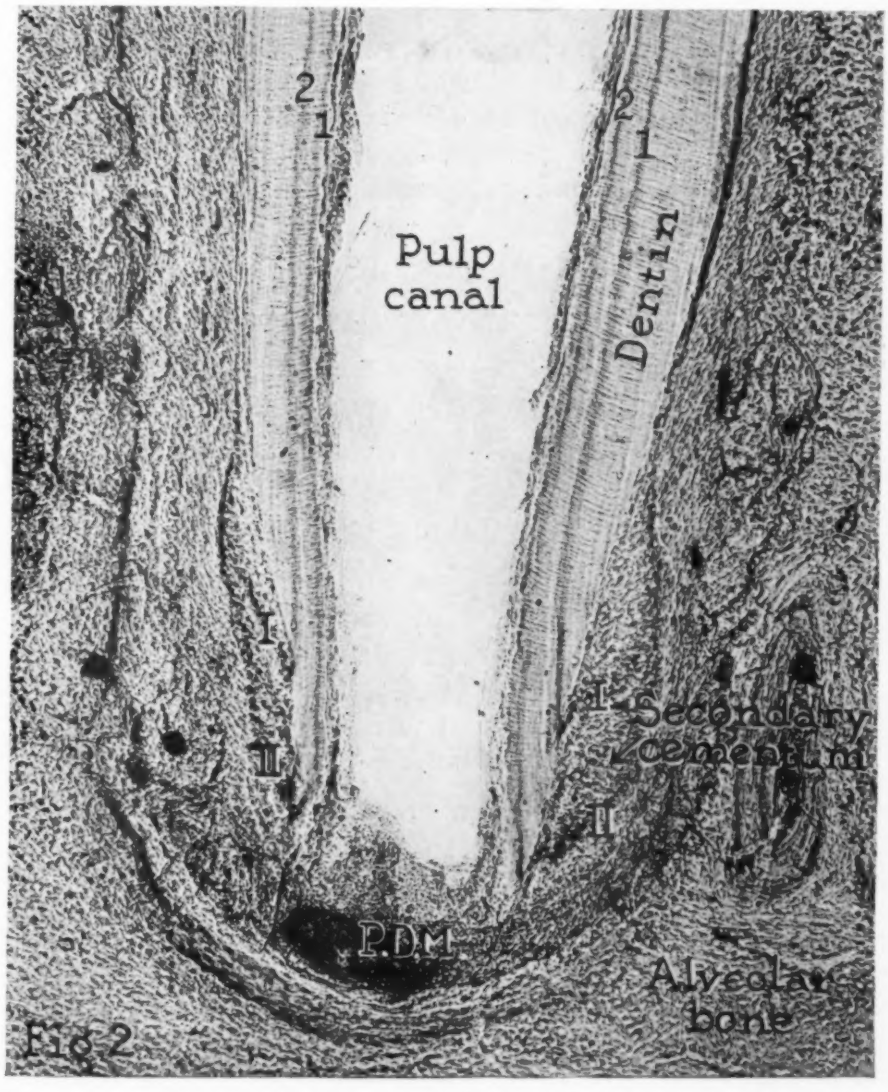

Fig. 2.—Photomicrograph of a ground section showing the distal root of the mandibular first molar of an albino rat which was given two injections of alizardin red S on the fortieth and fifty-fourth days of life, and sacrificed on the sixtieth. Note the alizarin red S effects in the dentine, *1, 2;* secondary cementum, *I, II;* *P.D.M.*, periodontal membrane. (98×.)

Chronology and Growth Pattern of Secondary Cementum.—Beginning with the fifth to the sixth week the chief portion of the new cementum is of the secondary type, which is cellular and contains embedded periodontal fibers (Fig. 2). Up to 12 weeks of age its increments meet the dentino-cemental junction at an

initial angle of 15° which increases to 40°. These increments thus follow a pattern which at first is a mirror image of the synchronous dentine increment. After the twelfth week, when the dentine no longer contributes to the elongation of the root (Hoffman and Schour[6]), the increase in root length results solely from the apposition of secondary cementum. The increments of the latter now take the form of inverted cones which cover the root apex at both the periodontal and pulpal surfaces (Figs. 2, 3 and 6F). The apex of the cementum cone now becomes the apex of the root.

Rate and Apposition of Secondary Cementum

The average rate of apposition of secondary cementum from the fifty-sixth to the one hundredth day was approximately 53 μ per week (Table VI). This rate decreases with age. It is much more rapid than the dentine rate that obtains during the corresponding periods (Hoffman and Schour[6]) (Figs. 2 and 5). The fastest rate of apposition for secondary cementum occurred between the sixth and seventh weeks and was approximately the same as the rate of root elongation at the time (Table VI).

Fig. 3.—Photomicrograph of a ground section of the apical portion of the distal root of the first molar of a rat which was given injections of alizarin red S at 63 and 77 days of age and sacrificed at the ninety-first day. Note the effects in the alveolar bone, *Al.B., 1 and 2,* and in the secondary cementum, *C, I and II. P.D.M.,* periodontal membrane. (98×.)

ALVEOLAR BONE

The alveolar bone grows by apposition, layer upon layer, and each red line is indicative of the incremental layer of bone which was growing and calcifying at the time of each injection. There is no indication of interstitial growth. The distance between two successive lines differs strikingly at different levels, indicating prominent gradients (Tables II and III). As a rule the incremental lines were continuous and extended along the crest, the wall of the alveolus, and the fundus (Fig. 5). They were interrupted in the areas where resorption had

occurred. The composite effect of the red lines resulting from the multiple injections of alizarin red S constituted the pattern of growth of the alveolar bone (Fig. 4).

Alveolar Crest.—The bone at the alveolar crest is apposed one cone or layer upon another in an occlusal direction (Fig. 5). Its rate of apposition (Tables VI and VII) depends upon the age of the animal, the particular tooth which it serves, and the physiologic forces, such as mesial drift and occlusal stress, which act upon the tooth or the entire segment. The crest bone grows faster than the fundic or the alveolar-wall bone.

Differences Between the Distal and Mesial Crests.—At the distal crest the fastest weekly rate was 190 μ during the second to third week, and graded down to 91 μ during the fourteenth week, while at the mesial crest the weekly rate was at first 160 μ and graded down to 71 μ per week. The differences in rates between the mesial and distal crests became more marked after the eighth week of life, when bone growth over the mesial crest had ceased along the periodontal surface, but became more marked at the periosteal surface. The portion adjacent to the periodontal membrane undergoes resorption during this period and part of the gingival portion of the mesial root becomes exposed to the oral cavity.

Fig. 4.—Photomicrograph of a ground section of the distal root of the mandibular first molar of an albino rat which was given eleven injections of alizarin red S between the fifty-sixth and ninety-first days of age and was sacrificed at the ninety-third day. Note the zones (see arrows) in the bone, *Al.B.*, and secondary cementum, *C*, which were demarcated by the injections. *P.D.M.*, periodontal membrane. (139.3×.)

The distal alveolar crest constitutes part of the interdental septum between the first and second molars, and arises entirely from the periodontal membrane. Unlike the mesial crest, it shows no resorption at its mesial surface, where apposition continues at a more rapid rate than on the mesial crest. The difference in the mesial and distal crests apparently results from the pull and pressure stresses exerted on the bone.

Interradicular Crest.—The rate of apposition on the interradicular crest approximates the rate at the distal crest. The most rapid rate occurs during the first five weeks (Tables VI and VII).

Fundic Bone.—The fundic bone grows slower than the alveolar-crest bone (Table VI). Each increment is flat or slightly concave and is apposed one upon the other in an occlusal direction, the last one being nearest to the periodontal membrane and to the apex of the tooth (Fig. 4).

At approximately the fifty-sixth day the fundic bone of the mesial root tends to grow more rapidly at the mesial corners than at the distal corners. The alizarin rings tend to converge toward the distal corner or are entirely lacking here as a result of bone resorption in this area. The maximum weekly rate was 54.3 μ, the minimum 6.1 μ (Table VI).

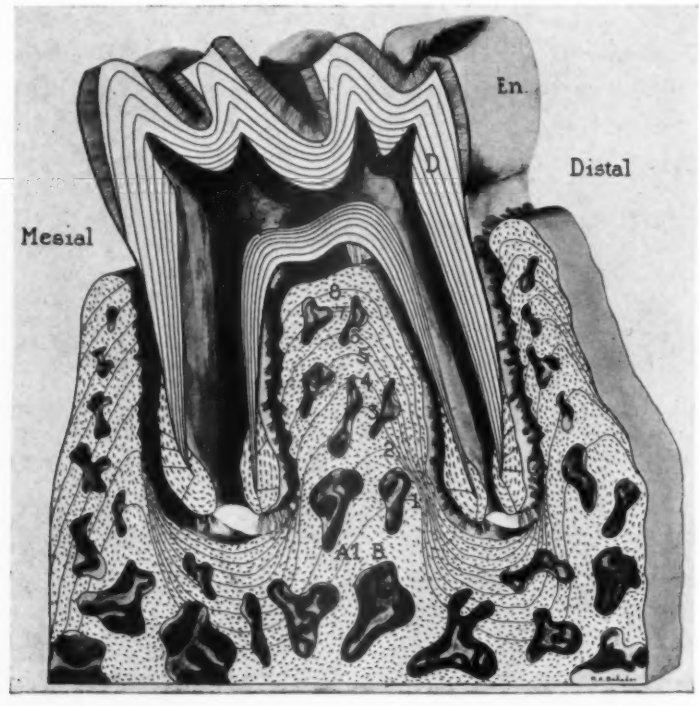

Fig. 5.—Semidiagrammatic, three-dimensional reproduction of the mandibular first molar and the surrounding alveolar bone in an animal 100 days of age. The time between the incremental lines of growth in the dentine, *D*, alveolar bone, *Al.B.*, and the secondary cementum, *C*, is approximately fourteen days each. The lines show the composite incremental effect. Note the gradient and the relative positions which the tooth attained from the first to the last injection, *1-8*. The resultant direction is mesioclusal. Resorption areas are indicated by the wavy outline of the border of the alveolar bone and the interruptions in the continuity of the incremental lines. *P.D.M.*, periodontal membrane; *En*, enamel. (11.7×.)

Alveolar Wall.—The daily rate of apposition along the walls of the alveoli was less than at the crest and fundus, and ranged from 25 μ to 0.6 μ (Table VII). The rate also varied considerably within any given period as a result of the alternating resorptive and appositional phases. For example, in the group of animals which were given ten to twelve injections between fifty-nine and ninety-three days, the interradicular bone adjacent to the distal root showed continuous active apposition, but the rate was not constant at all levels. It was greater toward the crest region than toward the apex. On the other hand, at the mesial alveolar wall next to the diastema, the surface of the crest facing the periodontal

TABLE IX

THE RATE OF ERUPTION IN MICRONS OF THE MANDIBULAR FIRST MOLAR OF THE RAT MEASURED BY TWO METHODS

	AGE IN DAYS	14-21	21-35	35-49	49-63	63-77	77-91	91-100	100-125	125-200	200-300	300-400	400-500
Average weekly rate of eruption in mm.	Obtained by measuring the change of position of C.E.J. in relation to fixed alizarin red S effects in alv. crest	844.1	588.6	161.4	144.9	110.9	90.5	81.7	68.4	44.0	26.8	21.1	13.4
	Obtained by adding rate of fundic bone apposition and rate of root elongation	835.4	594.1	157.9	138.2	104.3	93.7	83.2	68.7	43.1	26.5	20.2	12.8

membrane did not stain with alizarin but showed resorption (Table VII and Fig. 5). The crest was growing primarily from bone that was apposed by the periosteum which adjoins the diastema between the first molar and the incisal edge of the incisor. At the beginning of the apical third of this wall the alizarin red S lines tend to converge abruptly and then immediately diverge as they approach the depth of the fundus where they reflect a more rapid rate of apposition. This point of constriction suggests the probable site of the fulcrum of the tooth.

Persistence of Alizarin.—The alizarin effects in the compact bone as a rule are continuous lines. Most of them persist for thirty to forty days before they are resorbed; that is, before the bone becomes cancellous. Hence it was possible to study in a ground section the effect of an injection given thirty to forty days before death and utilize it as a point of origin and reference from which subsequent alizarin red S effects could be allocated and from which measurements, consequently, could be made. Each line thus served as a fixed point from which the eruptive process could be studied. The latter is discussed below.

PERIODONTAL MEMBRANE

Table VIII gives the measurements of the width of the periodontal membrane at different levels and ages. The width at the apical region and at the interradicular-crest level remains relatively constant throughout the first 500 days. This width was confined to a range of 125 to 135 μ at all the ages indicated. Measurements made from photomicrographs of given magnifications indicate a similar condition. The range is partly due to the discrepancy which results from sections which are not truly midsagittal. The measurements at other levels along the walls of the alveoli in general show the width to be an inverse function of the age.

RATE OF ERUPTION

There was no significant difference between the estimates of the rate of eruption that were obtained from the two different methods: (1) Rate of change of the position of the cemento-enamel junction in relation to the dated alizarin red S effects in the alveolar crest; and (2) the sum of the rates of apposition of fundic bone plus rate of root elongation.

The rate of eruption was greatest prior to the establishment of functional occlusion (thirty-five days) and decreased with age (Table IX).

The initial appearance of the tooth in the mouth at about the nineteenth day did not markedly retard the rate at which the tooth was erupting. However, when the tooth came into occlusion an abrupt retardation became apparent. Fig. 8 shows that the eruption rate assumes a line which is nearly parallel to the vertical axis up to thirty-five to forty days. Following this period the line assumes first a sharp and then a gradual curvature.

The eruptive rates graded from more than 800 μ per week during the first two weeks to approximately 13 μ per week at 500 days of age.

While our experimental data are limited to a period of 500 days, the alizarin effects in a 500-day-old animal indicated that eruption had not stopped

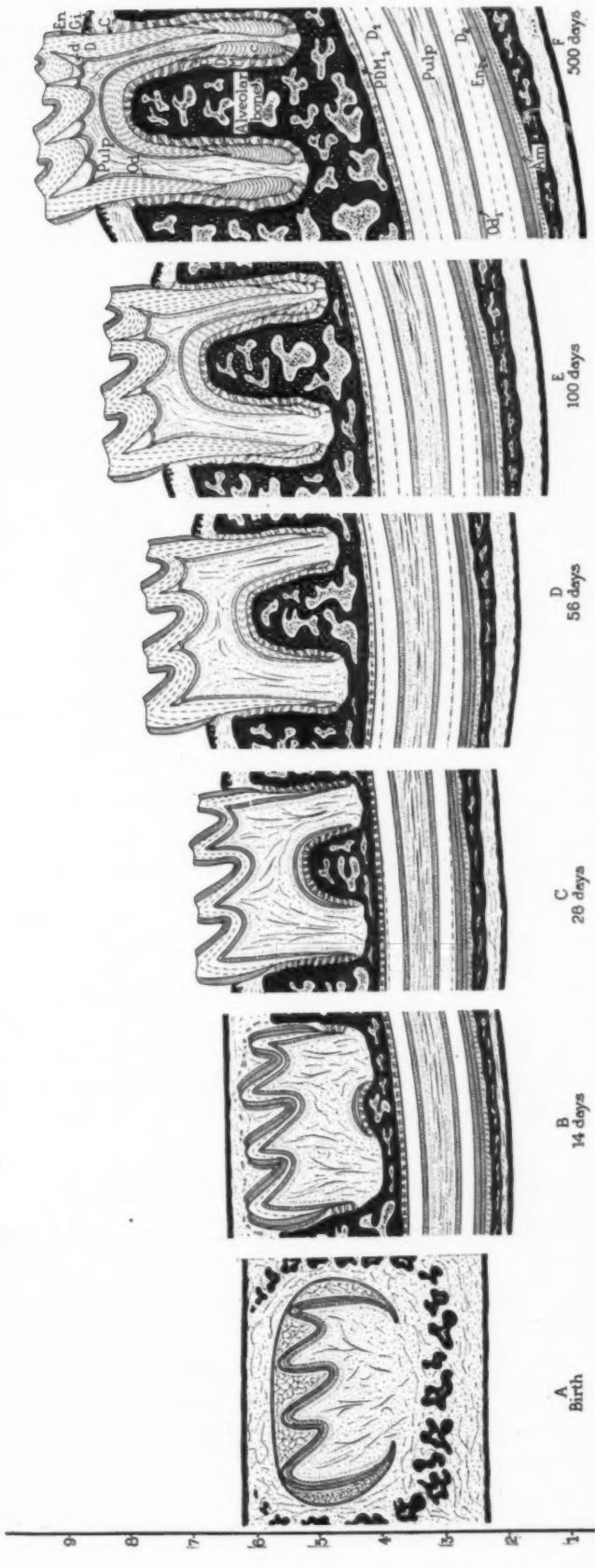

Fig. 6.—Semidiagrammatic reproductions of the mandibular first molar of the albino rat showing the relative positions assumed by the tooth from birth to 500 days of age. The mandibular incisor is included in the diagrams. Molar legends: *C*, primary cementum; *c*, secondary cementum; *D*, primary dentine; *d*, secondary dentine; *En*, enamel; *Gi*, gingivae; *Od*, odontoblasts; *P.D.M.*, periodontal membrane. Incisor legends: *Am.*, ameloblasts; D_3, cementum-covered dentine; D_2, enamel-covered dentine; *En*, enamel; *Od*, odontoblasts; *P.D.M.*, periodontal membrane.

but was still continuing. At 500 days there was still apposition of bone at the crest and fundic areas, and the root length was still increasing as a result of secondary cementum growth.

Duration and Path of Eruption

Eruption begins before the crown is completed and probably with the beginning of the formation of enamel and dentine, and continues throughout life. The records engraved in the alveolar bone by means of vital staining with alizarin thus chart the movement of the teeth and confirm the findings of Stein and Weinmann[10] on the physiologic mesial drift.

An analysis of the data found in anteroposterior sections and presented in Table VII and Fig. 5 shows that the mandibular first molar migrates both occlusally and mesially, and at the same time also tilts mesially. Buccolingual sections show migration in an occlusobuccal direction which proceeds not in a straight line but along an arc in which the crown tips lingually and the apex shifts buccally. This type of movement contributes to the lateral expansion of the face. The alizarin red S effects show the path of eruption to be a resultant of all the above directional movements.

DISCUSSION

Tooth Development a Continuous Process.—On the basis of the microscopic findings in animals up to 500 days of age and the roentgenographic findings in animals of more advanced age, we may conclude that tooth development which in its broader sense consists of growth, calcification, eruption and attrition, and the development of the supporting structures continues to at least 1000 days of age and in all probability throughout the life of the rat.

Quantitative studies of rates of activity and their chronology prove that the following processes, while proceeding at various rates, are continuous and are closely and harmoniously interrelated:

1. Eruption
2. Apposition and resorption of alveolar bone
3. Apposition of primary and secondary cementum and root elongation
4. Attrition
5. Apposition of secondary dentine

Long after the initial growth potential for the deposition of enamel and primary dentine is expressed, the supporting tissues and secondary dentine continue to be deposited. Thus ontogenetic development not only expresses phylogenetic development but continues beyond the latter in response to the physiologic demands of the individual organism.

Eruption.—Our data show that eruption is continuous and confirm experimentally Gottlieb's Theory of Continuous Active Eruption.[5] The continuous apposition of fundic bone shows that the root end is moving definitely occlusally and not centrally. If there were any downward migration, we would find resorption of the fundic alveolar bone instead of apposition. Since the periodontal membrane remains constant in width, the occlusal movement of the root should

be at least equal to the total amount of apposition of fundic bone. But measurements show that while the apposition of the fundic bone continues there is also an increase in the length of the root. It follows, therefore, that the actual occlusal movement of the tooth should be equal to the sum of the amount of root elongation and the total apposition of the fundic bone.

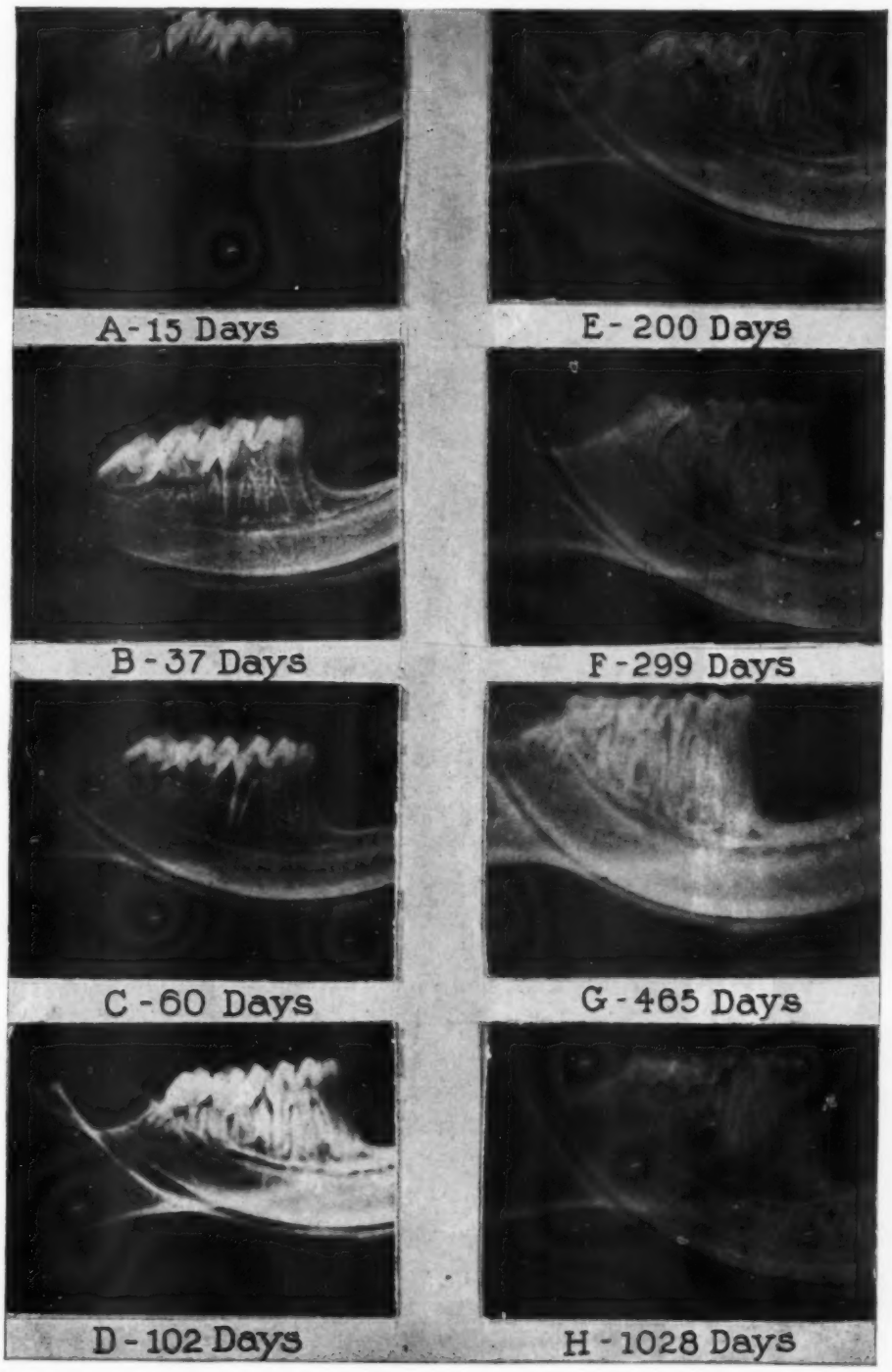

Fig. 7.—X-ray photographs showing the development of the mandibular three molars from 15 to 1028 days of age. (3.6×.)

In order to check this conclusion, another independent method was employed which consisted of measuring the distance that a fixed point of the tooth traversed in relation to another fixed level in the alveolar bone. The cementoenamel junction was used as the fixed point of the tooth. This point was chosen because it was easily recognized and was not lost with age. In the alveolar bone the alizarin red S ring of a given date as recorded at the alveolar crest was used for a fixed level. Since there is no interstitial growth a given alizarin red S effect could not be changed except by resorption. Before a given alizarin landmark would be lost, measurements were transferred to the successive alizarin ring of known date and corrected accordingly.

It is significant that measurements of the rate and total amount of eruption obtained by two entirely different methods gave the same results (Table IX).

Hertwig's epithelial sheath is intimately associated with and situated at the growing dentine end of the root. This epithelial sheath, therefore, is, as Orban[7] has pointed out, not growing in an apical direction. For the most part it moves occlusally, together with the dentine root.

Relationship Between Alveolar Bone Growth and Eruption.—Our findings indicate clearly that the normal alveolar bone growth is secondary to tooth growth, to tooth eruption, and to the forces exerted on the tooth. Despite the very rapid rate of tooth elongation and of eruption, the alveolar bone did not grow nearly as fast, either at the crest or at the fundus. For example, during the period from twenty-one to thirty-five days when the root was elongating at the rate of 540 μ per week and the eruption rate was 590 μ per week, the rate of alveolar bone growth was about 54 μ per week at the fundus and 168 μ per week at the crest.

On the basis of these findings we cannot subscribe to the view of Brash[2] and others that eruption is subservient to alveolar bone growth. For, if this were true, then alveolar bone should grow as fast as or faster than the rate at which the tooth is erupting. Brash's experiments with madder feeding (Brash[2]) were not carried out on a quantitative basis, and did not permit a quantitative dissection of the various factors related to eruption.

Apposition of Secondary Cementum and Root Elongation.—Our quantitative findings confirm Gottlieb's statement[4] that a considerable portion of the root of the molar of the adult rat is composed of secondary cementum. The rate of apposition of the latter is such that the total length of the tooth increases continuously in spite of the continuous shortening of the anatomical crown by attrition. This activity on the part of cementum is thus in excess of the amount necessary to compensate for wear. It is more than sufficient to keep pace with the continuous eruption which is responsible for at least some of the increase in the intermaxillary height of the face. The curve charting the rate of tooth elongation (Fig. 8) is a typical hyperbolic growth curve of postnatal development.

Attrition.—Attrition is continuous and compensates for the continuous process of active eruption so that the clinical crown remains constant in size. At the same time the extra-alveolar length or level of the tooth also remains constant in size, with the result that the occlusal and masticatory stress remains within the physiologic range of the tissue tolerance of the periodontal structures.

In an experiment in which the maxillary left molars were extracted at 21 days of age and the animal was sacrificed at 45 days of age, the mandibular left molars showed supra-eruption in comparison with those of the right side. Thus the expression of their eruption potential was favored. Furthermore, they showed no attrition and retained their infantile cuspal anatomy at their occlusal surface. The mandibular right molars on the other hand showed a beginning of the physiologic flattening of their occlusal surface.

The continuous process of attrition serves to compensate for eruption, to dissipate the impact caused by mastication, and to regulate the articulation. It is interesting to note that while the curves obtained by charting the rate of tooth elongation and eruption (Fig. 8) are typical hyperbolic growth curves of postnatal development, the charting of the rate of attrition results in a straight line between ordinate points. This possibly indicates that attrition is a mechanical process resulting from function and is independent of growth (degrowth).

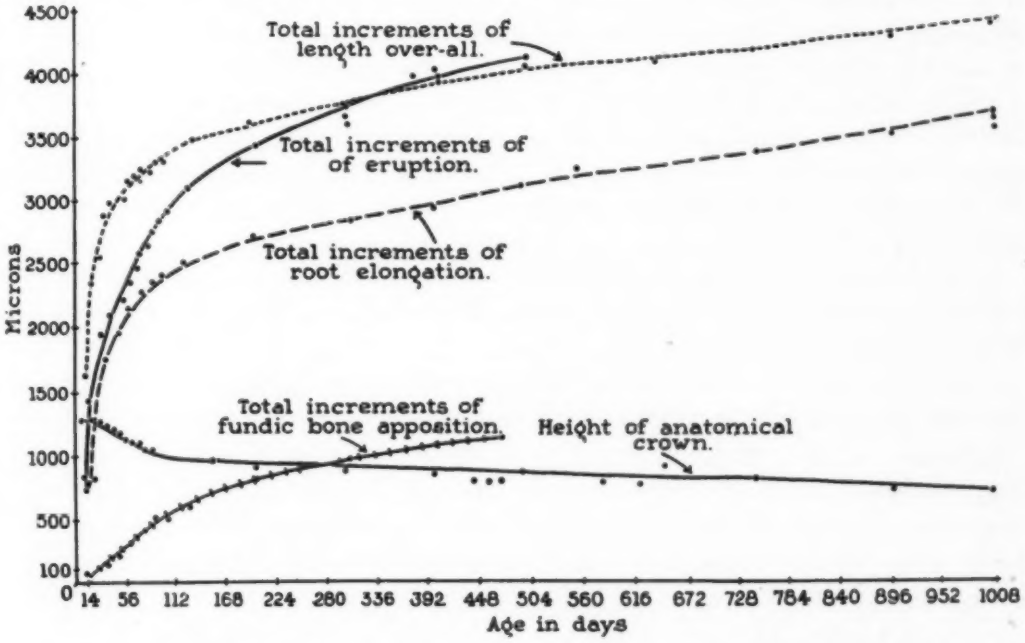

Fig. 8.—Graph showing total increments of growth, eruption, and height of anatomic crown of the mandibular first molar of the albino rat from birth to 1008 days of age.

Secondary Dentine.—Concomitant with the continuous process of attrition, secondary dentine is being apposed and the pulp recedes. The first appearance of secondary dentine synchronizes with the time when the opposing teeth come into clinical occlusion and is marked by a rate of apposition (Hoffman and Schour[6]) which is faster than the rate of the primary dentine which immediately preceded it. This initial high velocity of secondary dentine formation gives the characteristics of a secondary growth center. However, the formation of secondary dentine cannot be ascribed entirely to the process of attrition. Secondary dentine was found in the molars whose antagonists were removed prior to occlusion, and secondary dentine has been described in retained teeth.

The Constancy of the External Diameter of the Rat Molar.—Our data do not confirm the conclusions of Donaldson and French[3] that the external di-

ameters of the molars increase with age (Hoffman and Schour[6]), and do confirm the views of Wood and Wood.[11] The latter showed that the impressions of the rat molars which were taken earlier in life fitted the same teeth with mechanical precision at later periods.

The measurements of Donaldson and French[3] were no doubt correct but they apparently neglected to take into account the anatomy of the molar crown and the fact that the eruption of the rat molar is a continuous process. Their measurements represented the external diameters of the crowns at different levels and not at different ages. The maximal external diameter of the crown is at the middle third. Examination of Fig. 1 will show that the embrasure at the mid-coronal region is greater than at the cuspal surfaces. This level remains below the gingival margin for a considerable length of time until it reaches the oral cavity, as a result of the continuous attrition and eruption of the molar. Since Donaldson and French[3] measured the teeth of skulls at the occlusal level, their measurements were increasingly greater in older animals because in the latter the deeper levels of the crown that are of wider embrasure had become exposed as a result of the continuous and compensating eruption.

The increase in the anteroposterior length of the entire molar segment reported by these investigators is not due to external growth of the individual molars but a result of distal tipping of the axial inclines of the third molars and the shifting of the contact points of adjacent teeth concomitant with the path of eruption.

Physiologic Conditions in Rat Molar.—The rat molar presents ideal physiologic conditions in masticatory function and adjustments which are not common in the human teeth (Table II). After it has reached occlusion, the clinical crown of the rat molar remains constant in height as a result of the harmonious and continuous attrition which compensates for the continuous process of active eruption. Its extra-alveolar length, like the clinical crown, also remains constant in height as if the supporting tissues proliferate and elevate along with the tooth. The mechanical advantage of having a relatively short extra-alveolar lever and long intra-alveolar lever is increased with age because the root becomes increasingly longer as a result of the continuous apposition of secondary cementum (Table III).

Passive eruption in the sense of the clinical crown becoming longer as a result of the apical shifting of the bottom of the crevice has not been evident in our material. Gingival recession, downward growth of the epithelial attachment along the cementum, and atrophy of the alveolar crest are not common.

Gottlieb[4] refers to a communication from Erdheim that rats frequently lose their molars as a result of loosening or exfoliation. In our experience such a condition is not common. We did not find a single instance of loss of molars in the sixty-three rats which were stained vitally. Gottlieb points out that the loss of teeth was due chiefly to the inflammation produced by external injuries from animal hairs and strands of oat grain that became embedded as foreign bodies in the gingivae. The diet ingested by our animals was for the most part ground meal (Purina chow).

It would be interesting to speculate on the possible factors which make the rat molar a favored tooth as compared with the human teeth. The fact that

our rats have been living under very favorable conditions of diet and care and are derived from the Wistar stock may be an important factor.

The data obtained in this study are of special value in paving the way for a similar analysis in rats subjected to experimental interferences (Schour, Smith and Hoffman[9]) and other animals. The method of vital staining with alizarin red S is equally useful in the analysis of the growth pattern of the long bones (Aries and Hoffman[1]).

SUMMARY

The growth pattern of cementum and alveolar bone and the eruption pattern were studied in the rat molar by gross and radiographic analysis (166 animals from 13 to 1050 days of age) and by vital staining with alizarin red S (sixty-three rats from birth to 500 days of age).

The gross and radiographic findings were:

1. The clinical crown remains constant in height throughout life.
2. The extra-alveolar height of the molar remains constant in height throughout life.
3. The total length of the tooth increases with age, in spite of reduction of anatomic crown by attrition.

The histologic findings were:

1. Apposition of primary cementum continues throughout life in increments which are parallel to the dentino-cemental junction and which slow down with age.
2. Apposition of secondary cementum begins at 35 days of age. After 80 days of age it is entirely responsible for root elongation, which process is continuous throughout life.
3. The apposition of alveolar bone is subservient to the physiologic migration of the tooth. It continues in an occlusal direction at the crests and the fundi. The alveolar walls show appositional and resorptional adjustments to the movement of the tooth. The alveolar crest shows the most rapid rate of apposition.
4. Eruption is continuous throughout the period studied. It begins with the first evidence of calcification. The path of eruption is in an occlusal, anterior, and lateral direction. Fastest rates are found prior to 35 days of age. The rate of eruption equals the sum of the rates of root elongation and of fundic bone apposition, and decelerates with age. The rates of appositional growth of bone and cementum and the rate of eruption follow gradient effects. Quantitative data are presented and tabulated in the section of experimental findings.

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THE USE OF CHROME ALLOY

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A. The Possibilities of Chrome Alloy

Brooks Bell, Dallas, Texas

THE use of chrome alloy for orthodontic bands, attachments, and appliances has long since passed the experimental stage. The erroneous ideas that at first prevailed, and still are advanced in biased quarters, namely, that galvanic action in the mouth caused disintegration of chrome alloy appliances resulting in gingival irritation, that springs and attachments came off and bands came unsoldered or unwelded, that loss of elasticity in attaching springs to body wires occurred, have been proved, *in actual practice*, to be entirely false.

The truth of the matter is that the proper chrome alloy material offers greater elasticity, greater tensile strength, and greater resistance to corrosion and discoloration than any other material used in orthodontics; it is not a substitute for any metal, but is a metal far superior to any other available for orthodontic purposes. The fabrication of any type of appliance with the *proper* chrome alloy is as simple as the fabrication with any other metal and the technique of handling chrome alloy is as simple to master as that of any other material. The *proper* chrome alloy materials must be used, for some stainless steel or so-called chrome alloy products, is entirely unsuited for orthodontic purposes.

The following outlines of techniques are condensed in such a manner that the pertinent points of each are immediately evident. Bear in mind that to achieve these results, proper materials, instruments, and equipment must be used. With this thought we have freely mentioned the names of the manufacturers of the materials, instruments, and equipment which we have used successfully in our own practices.

At the Los Angeles meeting in 1938, I reported that during 1937, I had a total of 125 broken appliances. This meant an average of one broken appliance out of every 24.66 visits.

The following statistics compiled in our five offices during the months of March and April of 1940 again clearly indicate just how successfully chrome alloy appliances have operated in our practices.

The total number of office visits was 2,802.

Labials broken	9
Linguals broken	36
Springs (all types) broken	27

Read before the American Association of Orthodontists, Chicago, Ill., May 16, 1940.

Bands (Ant. & Post.) broken	17
Tubes (Buccal & Lingual) broken	9
Soldered linguals broken	20
Lingual Extensions broken	5
Hawleys broken	6
Intermaxillary hooks broken	17
Total breakage	146

This gives an average of one broken appliance to every $19\frac{14}{73}$ visits.

These statistics tell our story—lessened breakage means increased efficiency in the operation of appliances with the desired results attained at an earlier date.

B. New Ideas in Spot Welding Chrome Alloy

George Herbert, St. Louis, Mo.

1. *Welding* (The Rocky Mountain Metal Products Company "Tru-Spot" Welder is used.)

a. *Attachments to molar bands.*—Rocky Mountain Tru-Form Seamless molar bands are used.

Both the flanged buccal tube and the flanged lingual tube can be attached by welding. Anneal the flanges before welding to permit better adaptation to the bands. Then spot weld in only one spot at the gingival to permit alignment; align as desired and complete welding. Buccal tubes may be had in sizes .022 to .040, and lingual tubes in sizes .030 to .036.

b. *Attachments to anterior bands.*—To place the loop for rotating the tooth use annealed .015 wire. Using small nosed pliers make the loop and weld it where best suited to rotate the tooth, leaving approximately two millimeters of the wire on each side of the loop to weld to the band; then weld these ends in several spots.

c. *Welding springs to the lingual.*—In welding springs to the lingual use .016 wire, annealing about one-half inch on the end of the wire; then weld it to the .036 lingual so that when the wire is wrapped around the .036 wire it will take up all the annealed part of the wire. This leaves all the elasticity in the spring.

d. *Welding lingual locks.*—In welding lingual locks on the .036 wire use .016 wire. Anneal about one-half inch on the end of the wire and weld distal to the Ellis post, wrapping to the part not annealed; then bring the .016 wire under the end of the post and wrap anterior to the post. This end remains loose for adjustment and leaves all parts of the lock springy to hold the post in the tube.

e. *Welding loops on the labial wire.*—Anneal one-half inch on the end of a piece of .018 wire and weld to the labial wire about six millimeters anterior to the mesial end of the buccal tube. Hold the labial wire distal to this and, with round nose pliers about six millimeters in diameter, bring the .018 wire over the pliers and wrap it distal to the pliers. This end is left loose for adjustment.

2. *Repairing.*

Use tubing with an opening the same diameter as the wire to be repaired. Cut a piece about one centimeter long, placing the ends of the broken wire in this tubing about equally, and weld through the tubing several times on each end of the tubing. Then polish down.

C. Soldering Chrome Alloy

Mark Perrin, Topeka, Kan.

1. *Ellis Lingual Arch and Finger Springs*

The arch is made of .036 wire, the Ellis posts being formed in the usual manner. A great deal of breakage is eliminated by the process conceived by Dr. W. B. Stevenson; i.e., annealing the portion of the arch wire extending posterior to the post formed, grinding it down to 0.025, and forming the lock spring as a continuous portion of the wire.

The arch can be made by the direct or the indirect method, annealing being possible for adaptation in the latter method. Care must be taken not to anneal the arch to a red heat, permitting only a slight discoloration of the wire to occur and annealing only a small portion at a time.

In placing either the plain or recurved spring, flux and flow solder on both the arch and the 0.018 spring wire and then unite, care being taken not to lose the elasticity of the wire. A brief slight reddening will not cause a loss of resilient strength in the lingual, but will cause some loss of resiliency in the smaller wires. If such loss does occur, it can be restored by twisting the 0.018 spring wire on itself once.

2. *Labial Arch with Stop Springs and Intermaxillary Hooks, Using Seamless Molar Bands With Lingual Extensions*

Tru-Form Seamless bands are fitted on the first molars (the direct technique being used) and impressions taken with these bands in place. Wax is flowed inside each band before seating in the impression so the bands can be removed at will; the cast is then poured in regular plaster.

Lingual extensions of .030 Rocky Mountain wire are then soldered on, using Rocky Mountain flux and solder; these extensions engage the canines and second molars.

.032 round buccal tubes are soldered to each band and a labial arch of .028 wire is constructed. The stop springs are made of .022 wire and the intermaxillary hooks of .028 wire. Tubes and wires are of Rocky Mountain material.

3. *Arnold Coil Spring Lingual Arch for General Expansion*

Bands are constructed on the first molars (the direct technique being followed) using .180 by .006 Rocky Mountain Band material, which is lapped and spot welded to form the band.

A modeling compound impression is taken with the bands in place. The bands are then removed and a thin film of wax flowed inside so that they can be removed and replaced on the cast at will. Bands are then waxed in the impression and the cast is poured of ordinary plaster.

The entire arch is constructed of .032 Rocky Mountain chrome alloy wire. The left section is soldered to the band with Tru-Chrome solder and flux, en-

gaging the second molars and canines. This is done for greater stability, less breakage, and less tissue irritation. The anterior or expansion tube to fit the .032 wire is soldered to this left extension.

An .032 chrome alloy oval tube for the Ellis post is soldered to the lingual of the right band; the extension of .032 wire to the second molar is also soldered to this molar band. The right section of the expansion appliance is then formed in the same manner in which a lingual is made up, using the Ellis post and Stevenson lock to eliminate breakage.

The stop for the coil spring is formed with a drop of solder. Tru-Chrome Coil spring, .032 by .088, is used for the expansion pressure.

D. The Chrome Alloy Soldered Lingual

Leo Shanley, St. Louis, Mo.

Considerable difficulty has been experienced in soldering the lingual arch to the molar band. A welded re-enforcement before soldering has been used and found to be far superior to the plain soldered joint.

The Seamless Rocky Mountain molar bands are fitted directly in the mouth, after which the bands are removed and two rectangular Rocky Mountain wires .015 by .028 are welded to each band at the gingival margin on the lingual surface. These wires should be welded *only at the gingival margin* of the band, should assume a position on each side of the lingual groove of the tooth parallel to the long axis of the tooth, and should extend to the occlusal surface of the band.

With these welded wires in contact with the lingual surface of the band, the bands are replaced in the mouth. An impression with the bands in position is taken with Plastident.

The bands are placed in the impression and the entire surface of each band on the inside of the lingual is covered with sticky wax. The impression is then poured in artificial stone (Ransom and Randolph).

After the model is separated from the impression, the sticky wax is removed from the inner surface of the lingual of the band with a pen-pointed instrument. The entire lingual surface of the band will then be free from stone so that in soldering, the heat distribution will be uniform.

The lingual arch is then adapted to the model, after which the two rectangular wires which were welded at the gingival margin of the band are bent away from the band at the occlusal surface. The entire surface of the bands should be covered with flux (Kroloy).

The lingual arch is then placed back on the model and is seated in the lower point of the V which is formed by the band on one side and the flat wire on the other.

These wires are then bent with a band adapter around the arch wire, with the free end in contact with the molar band just below the occlusal surface. The arch wire will be held in position with these wires, after which the joint is soldered with .032 Kroloy solder.

E. A Twin Arch Technic in Chrome Alloy
(Following the Principles as Taught by Dr. Joseph E. Johnson)

George H. Siersma, Denver, Colo.

Orthodontists, particularly those using chrome alloy materials in the treatment of their orthodontic cases, will welcome the recent manufacture of attachments and materials necessary for the construction of the twin arch appliance. A complete choice of any orthodontic appliance is now available in chrome alloy.

This demonstration of the construction of the chrome alloy twin arch technique covers only those parts which differ essentially from any other chrome alloy appliance technique. Detailed appliance construction will be shown in the other sections of this clinic.

Essentially the differences between the construction of this twin arch appliance and any other chrome alloy labial appliance are the type of molar buccal tubes used, the anterior band attachments, and the labial arch itself.

The first step in the construction of the appliance is to fit and festoon the seamless molar bands accurately. With a small knife-edged file a mark for the buccal molar tube is made while the molar band is in place on the tooth. The .036 round buccal flange tube is used. With the file mark to aid in determining the position of the buccal tube on the molar band, one spot weld is made at the gingival, attaching the buccal tube to the band in such a way that if the tube is found to be attached in a wrong position, it can easily be removed. Now, before the tube is welded securely to the band, it is tried on the tooth to be sure that it is lined up correctly. Small adjustments can be made to the position of the tube without breaking loose the single weld. In lining up the tube, a short piece of .036 wire is placed in the tube while the band is in place on the tooth, and by raising or lowering the wire the tube can be pointed to the middle third of the first premolar tooth, or any other desirable position, depending on the type of malocclusion. This can be done only while the tube is tacked to the molar band with a single spot weld at the gingival. When the correct position is obtained, the band is removed from the tooth and the weld is completed.

The anterior bands may be fabricated from band material size .170 by .003 or seamless bands may be used. For both maxillary and mandibular incisors the twin snap channel brackets should be welded to the band in such a way that they are at right angles to the long axis of the tooth and in the center area of the band both mesiodistally and incisogingivally. All welded surfaces should be disked and polished.

The twin arch is made by first taking two lengths of tubing (outside diameter of .035½, inside diameter of .021) of length sufficient to extend from the distal of the molar tube to about the mesial of the first premolar. These will be for the end sections. A length of about six inches of hard .010 or .012 wire is doubled back upon itself in the middle. This wire is supplied in spools and as it comes off the spool it retains some of the curvature resulting from being wound on a spool. The wire should be bent back upon itself in such a way as to superimpose the curvatures. The two free ends of the wire are threaded through each of the two end sections. Then the two free ends of the wire are crimped several times with a pair of pliers. Then the tube sections are moved

to opposite ends of the doubled wire. They are kept from sliding off the wire by the crimps on one end and the loop made by the doubling back on the other end. To finish the arch, the two wire ends must be drawn into the tubes to the proper distance, which is the point where the arch has obtained its desired length. To accomplish this a special vise must be used similar to the one devised by Dr. Johnson and described by him in the literature. In arches where intermaxillary hooks are desired, they should be soldered onto the end sections before the wire is drawn. End sections can also be obtained already prepared with ferrule type intermaxillary hooks attached.

Where it is advisable to use Arnold coil springs to drive molars distally, size .035 by .010 should be used. These fit snugly over the end section tubing and a stop can easily be made by pinching the tubing with a sharp nosed plier. New stops can be made by simply making new pinches.

In placing the arch, the end sections are inserted into the buccal tubes and the twin wires in the anterior attachments. The twin wires are locked in the attachments with lock pins which snap in over them. It is sometimes necessary to squeeze the attachments slightly with a pair of Young's pliers to securely lock the pin. In cases where a tooth is too far out of line to lock the twin wires into the channel, a ligature tied from attachment to arch wire can be used.

The lock pins come in strips of four with a sufficiently long end to facilitate handling. Each pin is stamped so that it breaks off easily after it is snapped into place.

In the construction and manipulation of this appliance no special instruments are required other than the vise previously mentioned.

All the materials used in this demonstration are manufactured by The Rocky Mountain Metal Products Company of Denver, Colo.

PRACTICAL THOUGHTS ON ORTHODONTICS*

WILLIAM A. GIBLIN, D.D.S., MONTCLAIR, N. J.

AFTER a decade of practice in the field of orthodontics, attendance at numerous meetings, lectures, and courses, and perusal of many periodicals and books, one is impelled to take stock, meditate, and consider the various trends in this specialty. One becomes cognizant of the fact that there is a pendulum swinging between the mechanistic and the physiologic approaches to our problems, which has reached each extreme at intervals during the past fifty years. It is only during the middle path of its arc that the greatest advancements have been made. Several thoughts on questions which have arisen in my mind will be presented.

Of course our main endeavor is to bring about a definite occlusal relationship between the teeth of the maxillary and mandibular arches which will not only restore function but will be in harmony with the face and skull. Some of the main differences of opinion have been concerned with the latter. Angle has stated that the best harmony and the best balance of the face will be found in those individuals who have normal occlusion of the teeth. This does not mean that all individuals with normal occlusion will look alike. Nor does it mean that these individuals will be beautiful. However, the harmony and balance tend to a most pleasing appearance. The methods of producing this normal relationship of the arches are mainly those which employ mechanical appliances. Rogers and others have shown cases which by muscle training and the correction of bad habits have been corrected. However, the majority of us rely on appliances of one sort or another. This also has been the cause of debate between advocates of different appliance techniques. In addition, the regulating of these appliances is based on classification of the type of malocclusion. This, too, has led to controversy. As in other branches of the healing art where the ultimate aim is the prevention of various pathologies, we have investigators of the causes of malocclusion who also differ in their conclusions from, or interpretations of, observed phenomena—all of which confuses the humble practitioner and makes him wonder whether it is much ado about nothing. My views on some of these matters may be of assistance.

One of our first approaches to our problems is an understanding of the development of the face and jaws. This may be considered under the following methods:

1. *Evolutionary Method.*—In lower animals each type of face is associated with a definite pattern of behavior. So far we have not arrived at a definite pattern for humans. The various bones, their relationships and developments, can be traced through ages, as has been very capably presented by Gregory in his book, ***Our Face From Fish to Man.***

*Submitted as a partial requirement for a certificate of The American Board of Orthodontics.

2. *Anthropologic Method.*—This studies the variations in races as shown by skulls, and endeavors to arrive at general concepts of pure and hybrid racial types. This has given us the sagittal and Frankfort planes which have been used in measuring changes taking place during growth. Unfortunately, while these are easy to locate on a skull, we have to deal with living and growing individuals. Still, the ends mentioned in the first sentence, if obtained, would be of great assistance to us.

3. *Ontogenetic or Embryologic Method.*—This involves a study of the development of the growth of the head as a whole and of its various parts. It is essential for us to know the embryology of the face to understand the relationship between and the manner of development of the bones of the face and their associated structures. Up to birth we have the researches of the medical profession. After birth we have been able to do considerable research of our own. Hellman has made, in my opinion, the most painstaking and useful researches in this field until recently. Now the work being done by Broadbent, if it can be carried on, will give us more accurate information concerning the growth of both the face and the head, and most likely a method by which they can be correlated. If this can be simplified as to apparatus and cost, it will also be of decided aid to us.

4. *Genetic Method.*—This studies the heredity of facial characteristics, tracing through generations the results of homozygous and heterozygous matings with reference to particular features. We have never had proven that a mutation can be transmitted.

5. *Physiologic Method.*—This studies the chemical factors of the growth of the face, including those growth-stimulating substances that the embryo derives from its parents and those produced by its own endocrine glands.

6. *The Clinical Method.*—Certain types of faces are associated with low resistance to certain diseases, and this method seeks to discover the association between them.

In addition the face may be considered from a psychologic, criminalogic, or psychiatric viewpoint. It is important not to lose sight of the factors involved in the making of the human face and providing it with new functions in addition to those it performs in other living creatures. The changes which affect the jaws and teeth in the evolution of man are in large measure due to the growth of the brain and the enhanced significance of the face which higher powers of visual discrimination and understanding confer upon it. It is not only the most distinctive label of individual men, whereby their fellows recognize them, but also the chief instrument for expressing thought and feelings in social intercourse. Acquiring higher powers of vision was made possible by alterations in the position of the eyes, the reduction and transformation of the snout, and the reconstruction of the jaws with changes in number, form, and sequence of eruption of the teeth. The growth of the brain requires more time in humans, and during these years the eruption of teeth is delayed. If esthetic consideration and man's interest in faces played a part in the creation of a demand for orthodontics, the growth of the brain was chiefly responsible for disturbing the orderly process of uniform growth of the jaws from which many

dental troubles arise. As a background, the marked evolutionary changes which occur are:

1. Widening of the intercondylar diameter across the jaws associated with enormous expansion of the brain and partly with an increase in the width of the tongue.
2. A marked retraction of all front teeth with an upward and backward movement of the crowns of the mandibular incisor teeth, causing the procumbent incisors to assume a vertical position; a change in size, particularly diminution of the canine teeth which allowed their fitting into a smooth arch and the mandibular canines to occlude lingually to and with the maxillary lateral incisors and canine; and a change in the position of the roots and form of the rest of the posterior teeth. This, of course, results in our present short ovoid form of palate with narrow space between the canines.
3. The retardation in time of eruption of the third molar which sometimes fails to erupt.

The chin is the peculiar distinction of modern man. This was probably developed because the growth of the tooth-bearing part was restrained by delay in the eruption of the teeth. The alveolar part of the jaw does not increase in length from the seventh to the thirteenth year, whereas the basal part of the bone continues to increase in length, its growth being related to that of the body as a whole. Hence, the basal part of the bone is pushed forward beyond the alveolar part and the protuberance is the mental eminence. Its prominence is determined by the amount of use of the muscles of mastication and muscles of speech together with proper breathing. We may consider this as being expressed in the maxilla and associated bones by the greater prominence of the nose and the development of the aquiline form. The common saying, "As plain as the nose on one's face," is an unscientific recognition of the dominance of the nose in the human physiognomy.

In our particular field we are interested in the fact that the growth of the face may be measured in the three planes of height, width, and depth. Excepting the changes which take place up to the second year of life, the greatest increase in these dimensions occurs from the time of the addition of the first permanent molar to the eruption of the wisdom teeth. From the very beginning the greatest dimension of the human face is that of its width. This has been shown by studies in prenatal growth. In all subsequent growth there is an apparent effort of the dimensions of height and depth to catch up with that of width. This, though never accomplished, is closely approached. To attain this end the growth in the three planes becomes unequal with the result that increases in size are accompanied by changes in proportion. Thus, as the face grows larger, it becomes absolutely and relatively longer and deeper, the former exceeding the latter. Growth changes can be measured by time and dimensions as Hellman has shown in his studies, using as the time factor a scale classification of six phases in the development of dentition, and numerous anthropometric measurements for the dimension factor. I do feel that the recent work of Broadbent will supplant these because of greater accuracy with his cranio-

metric measurements and more detailed records of the growing child. This should give us a more definite picture of just where and at what comparative age growth should be occurring naturally. We should also realize that the normal growth of the nose, particularly the septum, depends on the normal growth of the associated bones, the ethmoid and maxilla, with the vomer contributing slightly. Deflected septums from above downward are very often associated with maldevelopment of the maxilla due to mouth breathing. Deflections from the anterior to the posterior are rather the result of accidents. All of the above, I feel, is a brief account of what we should know or think of in regard to the development of the face and jaws.

With this foundation we next come to diagnosis, which includes classification and etiology. I still feel that up to the present time we have no better system of classification than that given us by Angle. Most men who argue against this method claim that Angle uses the fact that in normal occlusion the mesiobuccal cusp of the maxillary first molar occludes in the buccal groove of the mandibular first molar, and that the mesiolingual cusp of the maxillary first molar occludes in the lingual pit of the mandibular first molar, as his "Key to Occlusion." They claim that he considers the position of the maxillary first molar as being constant. Many investigators have shown that this first molar erupts distally to the key ridge, and that through growth it gradually moves forward until it is mesial to this ridge. If there should be a loss of temporary tooth structure in advance of this tooth, it will show quite a forward drifting. Therefore, they claim you cannot use a movable part as a basis for classification. In addition to the first molar, Angle also used the fact that the maxillary canine occludes between the mandibular canine and first premolar. This is a check if there should be forward drifting of the first molar. While there is no question that the maxillary first molar moves mesially if there is normal occlusion, the mandibular molar also moves mesially in proportion. In fact, the completed cases of these men exhibit the above relationships. Of course, one who is specializing in this work is supposed to be trained to correlate the arches and jaws to the skull and cranium. No case should be classified from a set of models alone. One should also be able to distinguish micromaxillary and macromaxillary growth. Retroversion of either jaw should also be recognized. A careful study should enable one to classify any case. Most of the opponents of Angle's classification use the gnathostatic method of Simon. The author himself admits his orbital plane is based on a fallacy, but claims that we can get a more accurate diagnosis because one is able to correlate and measure the distances of various parts of the arch from the orbital and sagittal planes. In regard to the latter, what can we do about it after we know it? If one side of the maxillary arch is closer to the orbital plane, it may be due either to occlusal interference or underdevelopment of the maxilla on that side opposed to overdevelopment on the opposite side. Which of these last is to be used as a guide? With one side of the arch farther from the sagittal plane, the same question arises, which shall be used as the normal? The human skull is definitely and markedly asymmetric. It is not a question of the bones of the individual crania differing from a symmetric type, but the type crania itself is asymmetric. Some dimensions of the bones have dominance on the right side, some on the left side, but

on the whole the right side for size has dominance over the left side. If, as all evidence points, normality is shown in the main by asymmetry, why should we use a system of diagnosis which uses symmetry as its basis? And if we do, as I mentioned before, how are we going to determine which part to use as a standard for planning the movements to be accomplished? To consider Simon's orbital plane, we should realize that the mean position of Simon's orbitale relative to the dental arches is not constant from the fifth year onward. Its position in correctly occluded children of the same age shows variations to exceed sixteen millimeters, according to two most able investigators, Linden and Harth. Also, the mean position of the orbital plane, though near the canine tip in the four-to-eight-year period, is between the canine and premolars in the teens. The degree of constancy originally postulated by Simon is unattainable in any two points, one of which is on and the other above the dental arch. Broadbent has also proved the inconstancy of the orbital plane which is most conclusive, as his measurements and points are taken from the living child by both craniometric and anthropometric means. Other men have advocated the use of the "key ridge" as a landmark for classification of malocclusions, but since it is very difficult to have two men locate this ridge in the same place, this method fails in accuracy. Since the teeth have been shown to move gradually past this ridge in growth with normal occlusion, this classification would have to change with the age growth of the patient. After thoroughly comparing these various methods, I prefer to use Angle's system, with Lischer's terminology as the basis for my classification. Unless the researches of Broadbent bring forth a simpler and more accurate one, I will stay with my choice.

Hellman has given us face mask charts by means of which we can classify an individual within a given mean deviation. He has also set up a schedule based on the age and sequence of loss of temporary teeth and the eruption of permanent teeth as they should occur normally, and in addition has charted how deviation from this occurs in malocclusion cases. Todd, in his *Atlas of Skeletal Maturation,* has given us a means of classifying bodily maturation by skeletal age. If any individual is two years behind his scheduled age as shown by hand pictures, then that individual probably needs glandular therapy. He has stated we should be wary of retardations particularly. When this occurs, it is usually disregarded by the parents and physician, particularly if the deviation increases imperceptibly during the late teens. If, however, the inhibition of progress in maturation is sufficiently marked in the grade school period to be apparent, the chances of restoration during adolescence are very slender. To have hope of success, correction must be started before the tenth year. Maturation, like growth, is a complex phenomenon which depends largely on physical fitness. To be able to place an individual in a certain age group, either actual or skeletal after classifying the malocclusion, gives us a picture of the changes to be expected naturally during the coming years, and if we correlate this with our knowledge of how the face develops normally, we will be able to plan our treatment.

In a discussion of etiology we come into a field in which those who are interested in preventive orthodontics are very much concerned. The best presentation of this subject has been given by Dewey in Chapter IV of the fourth

edition of his book, *Practical Orthodontia.* I would recommend the repeated reading of this chapter. The question of the part heredity may play has been the subject of many papers. Nowhere have I been able to find definite or rather scientific evidence to prove that any malocclusion is inherited. The theory of large teeth and small jaws being inherited has failed to stand up under biologic research as have other conditions described as family traits. Some men contend that identical twins would have the same types of malocclusion, and numerous case reports have been presented. Their statements are based on the clinical evidence that certain identical twins showed a similar type of malocclusion, but those similarities were no more numerous or striking than similarities which could be found in children who were not twins, or even found among children who were in no way related. Gregory in his book, *Our Face From Fish to Man,* states: "Except in the case of identical twins, no two persons will carry the same hereditary factors affecting the growth of the jaws and face while even in the case of identical twins the nutritional factors can hardly be the same, especially after birth. The resulting diversity in form and appearance is bewildering and to some extent conditioned by the same law of chance associations of hereditary and environmental influences." It is a proved fact that certain evolutionary factors and conditions under which individuals live will produce a certain type of malocclusion regardless of any inherited relation between the individuals. Children suffering from the same acquired conditions will develop the same type of malocclusion. It has been demonstrated in lower animals that certain deformities can be produced by variations of diet. These deformities certainly would not be attributed to inheritance under those conditions. I wonder if this idea has been used as a good way to explain failures. The other causes of malocclusion—local and general—congenital and acquired—are too generally accepted to be discussed here. Preventive orthodontics can only be concerned with acquired causes and local causes after birth.

Now that we have diagnosed our patient against the background of development, we reach the stage of mechanical interference. I hold no brief for the man who is always changing his types of regulating appliances with each new (?) technique to be advanced. The question mark is used because in going over a history of orthodontics, one can find a forerunner of all types of appliances in use at present. Any appliance which does not allow a physiologic tooth movement should be discarded. The purpose of an orthodontic appliance is to exert pressure upon the teeth and thereby create cell metabolism, and as a result of cell metabolism, activities are produced which change the supporting tissues. This change is brought about by growth changes which result in a change in the form and size of the maxilla and the mandible. We must also realize that bone changes take place in regions far removed from the immediate application of pressure on the malposed tooth. Kronfeld has shown that pressure applied to the teeth has produced changes from the condyle to the anterior portion. Oppenheim has shown that bone develops in spicules in response to mechanical stimulation and that these spicules are laid down in a comparable position to the direction of the force applied. This histologic fact is something which has a great bearing on the problem of retention. There are some appliances being used, such as the pin and tube appliance, which are so

constructed that they produce a bodily movement of the tooth which would necessarily produce a decided bone change; and if the pressure were not too great, considerable bone development would occur. However, clinical experience showed that after teeth were moved in such a manner, it was necessary to retain them for a considerable period of time, regardless of the fact that there seemed to be a normal amount of bone development. The necessity for retaining these teeth can be understood when we realize that the bone which had been developed by using such an appliance had developed as a result of mechanical force and consequently the spicules were not laid down in such a manner as to support the teeth against the stress of mastication. It has also been found that the best results can be obtained by applying a light pressure over a considerable time, as the light pressure more nearly approximates physiologic bone growth. Under this condition the tooth is moved more by cell metabolism produced by the mechanical force. In this procedure it is not necessary to use a mechanical device to retain the tooth after it has reached its proper position. Excessive pressure can be judged by excessive redness of the tissues and soreness of the teeth. No pressure should be used which causes soreness in the teeth or supporting tissues over a few hours. Add to these facts the requisites of efficiency, cleanliness, and inconspicuousness, then one is ready to choose from the mechanical appliances in use that which he deems best fills these demands. Once having chosen, he should thoroughly master its construction and manipulation and give it as complete a test as possible. In my opinion, the combination of the lingual and labial appliances as advocated by Mershon, Lourie, Oliver, and others, best fills all requirements. It is essential that any type of appliance used must be one which will allow the greatest possible freedom of growth and development during the period of treatment. Any style of labial or lingual appliance which lashes the teeth together rigidly is an improper one because it interferes with proper growth and development.

Patients come to us for correction of some deformity, and it is our duty to do the utmost in our power to aid them. I do not hold with men who boast of having fifty per cent failures in their practice. I do not expect to secure an ideal occlusion in every case treated, but if I can secure a functional occlusal efficiency in harmony with the individual, that is my aim. I feel that this must be accomplished in over 80 per cent of cases treated, or we are decidedly at fault. Except in adults or cases which exhibit definite over-development, I do not believe in extractions. How men can claim that the apical base will not support the number of teeth in the arch of a child is beyond me. How can the growth possibilities of any child be foretold? In any case where the procedure is to move teeth, distally particularly, I advise extraction of the third molars at the earliest age the patient can tolerate it. Rotations, to my mind, are the movements which demand the most retention.

The question of root resorption is another bone of contention. As far as researches have shown, this phenomenon occurs equally in treated and untreated cases. Those cases which show root resorption before treatment should be very carefully regulated. We do know that during treatment some cases show root resorption, but the reason for it is obscure. Experimentally by excessive pressure we have produced root resorption in animals. Just what harm root resorp-

tion may do has never been shown or proved. It has not harmed the vitality of the teeth or rendered them more susceptible to decay or led to their early loss. However, as it is undoubtedly pathologic, we should try to avoid causing it during treatment.

The trend, as I view it, will be more to a thorough grounding in all sciences connected with the human body as outlined under the methods of studying growth and development of the face, and then a complete mechanical technique to carry out treatment. We should be in the same position as a surgeon. We should know enough to confirm a diagnosis and to make one. However, we are both users of surgical procedure. Most of our time should be used in perfecting our procedure or mechanical therapy. We should rely on allied sciences for researches on our problems. We should ask freely for assistance, and support research as much as possible. There are many secrets of nature we are yet to learn, but that is no reason to blame our failures on the unknown. Rather they should be a stimulus to us to seek to unlock the door to these secrets, or at least show the way to those who will succeed us. There are still other thoughts, but I hope these will serve as a spur to fresh views on our common profession.

85 Park Street

REPORT OF THE AMERICAN BOARD OF ORTHODONTICS

Since the meeting last year in Kansas City, the routine activities of the board have proceeded in the usual manner, no serious difficulties of any kind having been encountered.

For the last year or two the board has given a great deal of consideration to the advisability of changing the name from "The American Board of Orthodontia" to "The American Board of Orthodontics." Many authorities were consulted, and it was finally decided that the latter terminology was the more correct and in keeping with modern usage both in dentistry and medicine. It had already been ascertained that the name could be changed with very little trouble or expense. A fee of one dollar was charged by the Secretary of the State of Illinois for having the change of name recorded and there was some little expense attached to our inability to use a few blank certificates which were still on hand. The necessary details were all completed some time ago, and it is believed the change will meet with the approval both of the American Association of Orthodontists and the profession generally. This contemplated change in the name was contained in the report to the American Association of Orthodontists in 1939 in which we stated that, "With the approval of the American Association of Orthodontists the board will complete the necessary steps required to make this change." The report was approved by the American Association of Orthodontists and accordingly the board proceeded with the details as specified above.

At the request of the New York Society of Orthodontists, a brief paper describing the work of the American Board since its founding in 1929 was read before that society and later published in the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY. The object of the paper was to familiarize both orthodontists and general practitioners with the objectives of the board, and to supply them with information that would be useful in preparing applications. Such information, while always available through the secretary, has not been as well disseminated in the past as it might have been, due possibly to the hesitancy of the board to appear in the role of solicitor. The board contemplates the preparation of a similar paper to be published in the *Journal of the American Dental Association* designed not only to give the above information but also to give general practitioners other information which they might find useful in referring patients to orthodontists particularly in distant parts of the country.

In order to clarify and standardize the requirements for certification, the Board has adopted the following resolution:

WHEREAS, the American Board of Orthodontics has, in the past years, certified applicants who had been in the exclusive and ethical practice of orthodontics for more than fifteen years on their records only, and

WHEREAS, in a number of instances this action has resulted in criticism of the American Board of Orthodontics, and the purposes for which it was brought into existence,

Therefore, be it resolved, that this custom be discontinued, except where such records have fulfilled the following requirements:

A. The ethical and exclusive practice of orthodontics for a minimum of fifteen years, and in addition, one or more of the following:

B. An adequate number of contributions made before recognized dental, orthodontic, or allied organizations, said contributions having been published in the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY, *The Journal of the American Dental Association*, or other journals of satisfactory standing.

C. The applicant is the author of a textbook of dentistry or closely allied fields in at least its second edition.

D. Outstanding teaching or educational experience.

E. Where, in lieu of these contributions to the literature of orthodontics or allied sciences, such candidates submit a minimum of five case reports and a satisfactory thesis upon some phase of orthodontics.

It will be interesting to the members of the American Association of Orthodontists to know that the example of the American Board of Orthodontics is being followed by other specialties of the dental profession. Two or three of the other specialties either have completed organization of a certifying board or are in process of so doing. In addition to this a movement to inaugurate an advisory board, to be composed of representatives of certifying boards already established or in process of organization, was inaugurated two years ago. This board was actually set up last year at Milwaukee under the patronage of the American Dental Association. The American Board of Orthodontics is glad to cooperate in such a movement since noting the advantages already demonstrated by a similar organization which already exists in medicine composed of representatives of the eight or ten certifying boards which they have already established. Such a board provides opportunity for coordinating the activities of all of the specialties represented and should be as helpful in the dental profession as it has already proved to be in medicine. It might also be interesting to know that many hospitals today limit their appointments to men who have been certified by their own specialty examining boards.

The Ketcham award committee has, as in the past, made a very just and, I believe, a popular selection this year. From the many outstanding orthodontists whose contributions are more than adequate to entitle them to this honor, the committee's decision this year was that the award should be made to Dr. George W. Grieve of Toronto, Can. Here is a man who has been a faithful supporter and contributor to orthodontic literature. He has worked indefatigably to try out important methods of practice and has developed original concepts regarding occlusion and appliances which he has eloquently and fearlessly presented to the profession. This was referred to more fully during his presentation to the society for this award and in passing we rather regret that this presentation could not have been made, as it has been on other occasions, during a session which could be attended by guests as well as members of the association.

The work of the board this year, previous to and during this meeting, has been exceptionally heavy, and the secretary has not found time to prepare an exhibit from the large amount of material available. While this is not a strictly annual custom, such an exhibit probably will be presented next year as it was done last year in Kansas City, eliciting much favorable comment. We are happy to report, however, that the board received a request for an exhibit by the American Academy of Pediatrics to be coordinated with their own child exhibits at a three-day meeting which was held in Washington, April 4, 5, and 6. This request came through Dr. Erikson, president of the Washington-Baltimore Orthodontic Society, who was asked to supply some orthodontic material, and he reports that the exhibit created considerable interest there and was very well attended.

Up to the present time there have been issued 178 certificates, and there are now thirty applications before the board to be acted upon during this meeting. The genuine enthusiasm which most of the applicants exhibit in the preparation of their presentations is very encouraging and makes the rather arduous work of the board a pleasure.

Respectfully submitted,

H. E. Kelsey, President

REPORT OF THE PUBLIC RELATIONS COMMITTEE

Federal Activity.—During the year, the Federal Government has conducted proceedings of prime interest to the dental profession. The Inter-Departmental Committee, created at the instance of the President to study the National Health situation, has conducted additional hearings on the subject with special reference to medicine and dentistry. These investigations have had the appearance of being designed to promote certain legislation which would lead to regimentation under Federal and State compulsory and voluntary health insurance systems.

While these gave rise to new fear among medical and dental leaders who believed these actions were the forerunners of the further encroachment of the Government upon private enterprise, it was soon dispelled by an announcement by the President, that the Wagner Health Bill was prohibitive on account of its terrific cost.

A similar bill, however, has been introduced and is sleeping in Committee and will not under all probabilities be aroused at this session of Congress.

It should be pointed out, that the past year ushered in a new high record on the number of dentists being absorbed by government agencies.

Educational Standards and Legislation for the Specialties.—We are glad to report that widespread interest has continued to manifest itself in establishing more equitable and uniform educational standards to qualify for practice of the specialty.

The different organizations representing the specialties are continuing an earnest study of this program. They are slowly but surely moving forward with the idea of setting up systems of certification by their own group and licensure under State Dental Acts.

The proprietary schools are continuing to fall into disrepute and out of existence under this higher concept of graduate training for practice of the specialty.

A large number of the universities with dental schools are now providing one- and two-year courses of scholastic training, leading to a certificate of proficiency or a Master's Degree. Some of these universities are providing short courses of postgraduate instruction. Nearly all of them require the applicant to have a commendable orthodontic background to gain admission to the short courses. This is, of course, as it should be if reasonably adequate educational standards are to be established and maintained.

The Advisory Board for the Dental Specialties whose membership is made up of representative committees from all the major specialties and the Council on Dental Education, the National Board of Dental Examiners, the American College of Dentists, and the American Association for the Advancement of University Education in Dentistry is now completely organized and functioning.

This organization recommends that the preparation for specialists in dentistry after 1944 should be a Master's Degree or equivalent earned under a graduate school of a university and devoted to appropriate subject matter, plus three years in the practice of dentistry.

A committee to study legislation regarding dental specialties was appointed. The Committee is Dr. Leuman M. Waugh of New York City, Chairman, and Dr. William M. Bell of Asheville, N. C., and such other person or persons as the Chairman may desire.

It is at present acting as an independent organization with the purpose of ultimately becoming affiliated with some parent body, such as the Council on Dental Education.

The Advisory Board for the Dental Specialties should do much toward clarifying and coordinating the activities of these several groups in the fields of education and legislation.

The National Association of Dental Examiners is rightly showing a keen interest in licensing systems for the dental specialties. Many members of State Boards of Dental Examiners are of the opinion it is the most logical method of solving the problem.

Michigan may be added to the list of states now having amended their Dental Acts to provide a licensing system for the specialties. There are now four states which have adopted this system, namely: Tennessee, Illinois, Oklahoma, and Michigan. Leaders in education and legislation in other states are giving the subject serious consideration.

The Chairman of the Public Relations Committee was invited to attend a joint conference with representatives of the Independent Dental Journals and the American Association of Dental Editors at Milwaukee in July. It was the second conference held by these two groups to discuss business policies in connection with the advertising of commercial products and services. It was rather significant to find that the American Association of Dental Editors did not have a code of ethics to regulate the conduct of its journal members in the field of dental advertising. It is to be hoped that through a more friendly and united effort they may be able to formulate standards for business practice which will be reflected in better understanding and greater safety for the profession and the public.

Orthodontic Laboratory Advertising.—Both groups agreed that orthodontic laboratory advertisements should be classified as unacceptable principally due to the fact that they automatically engage in providing unreliable diagnostic and treatment services.

Public Relations Bureau.—During the past year the Public Relations Committee has devoted most of its time and energy toward establishing the Bureau and a program of activity.

The Board of Directors and Director Dwight Anderson have worked most diligently and faithfully and deserve the highest commendation of the association.

We feel the Bureau of Public Relations has been functioning in an admirable fashion. We are, however, mindful of the fact that we have been pioneering a large and unexplored field of orthodontic education and the reactionary effects of the past year's experiments and performances must necessarily serve as our best guide for designing its future cause.

A general report from the Director of the Public Relations Bureau to the Executive Committee is made a part of this report, together with a financial summary from the executive committee as follows:

REPORT OF PUBLIC RELATIONS BUREAU, AMERICAN ASSOCIATION OF ORTHODONTISTS,
MAY 1, 1939, TO APRIL 30, 1940

May 1, 1940

To:

Dr. T. W. Sorrels, Chairman,
Executive Committee,
Oklahoma City, Okla.

From:

Dwight Anderson, Director,
Public Relations Bureau,
292 Madison Avenue,
New York, N. Y.

Administration.—The wisdom of the unusual structural committee set-up for the first year of public relations work has been amply demonstrated by experience. Dr. Sorrels, as chairman of the executive committee, has kept in close touch with every detail through personal correspondence with Dr. Nicolai, as chairman of the Board of Directors, and Mr. Anderson as director of the Bureau. The file of this correspondence is voluminous. Thus the Executive Committee, widely separated geographically, has been the policy-fixing authority, while details of administration have been delegated to the Board of Directors, all located close to the office in New York, where Mr. Anderson's activities have been conducted.

Meetings of the Board of Directors have been held at the Columbia University Club in New York City, with the exception of a week-end meeting at Mr. Anderson's summer home at Center Moriches, L. I., and a meeting at the New York Athletic Club. The dates of meetings since the establishment of the Bureau are as follows: May 15, July 25, September 11, October 3, November 13, December 14, 1939, February 1, 1940, March 26, with the final meeting set for May 2.

The Executive Committee consists of T. W. Sorrels, Oklahoma City, Okla., Chairman; George A. Barker of Seattle, Wash., and Frank Nicolai of Brooklyn, N. Y.

The Board of Directors consists of: Frank Nicolai, Chairman, Norman L. Hillyer, Henry U. Barber, Joseph D. Eby, Sidney E. Riesner, J. A. Salzmann, Leuman M. Waugh, and Glenn F. Young, all of New York City.

Publications.—The Public Relations Bureau has assisted in the editing and approval of the final copy for two articles in popular journals, "Make Way for Even Teeth," in the September, 1939 issue of *Parent's Magazine,* and "Orthodontics for Adults" in the Fall 1939 issue of *You.* Another article is in preparation for *Life and Health,* by Dr. B. E. Lischer, Dean of the School of Dentistry, Washington University, St. Louis, Mo.

Communications have been mailed to the editors of fifteen publications of general popular circulation, requesting that our public relations bureau be consulted when material regarding orthodontic care is in preparation in their offices.

Distribution of Reprints.—Reprints were made of "Make Way for Even Teeth" to the number of 50,000 copies. They were offered to the membership of the Association and to special lists of names at a price to cover postage, printing, and overhead. The New York Society of Orthodontists purchased 10,000 copies, which were distributed free on their order to persons and organizations requesting them but who had no appropriation for making payment. Many inquiries were received from such sources, and we were thus able to supply educational material where it was needed and would be valued, which otherwise it might have been impossible to do, with a limited budget to meet an unlimited possible demand. The entire stock of 50,000 copies was distributed.

The Bureau purchased 1000 reprints of the editorial by Dr. Pollock entitled "School Hours and Professional Service for School Children" which appeared in the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY for December, 1939. These were included in the mailing of Bulletin No. 2, and the stock remaining was distributed in response to requests thus stimulated.

The leaflet, "Facts About Orthodontics for Health Workers" was made available on April 1 to the membership of the Association as well as to previous purchasers of "Make Way for Even Teeth." A total of 100,000 copies were printed. Letters concerning this document, with a sample and price list, were also sent to selected lists, including 585 members of the American Association for the Promotion of Dentistry in Children, and 500 tuberculosis associations having paid secretaries. The New York Society of Orthodontists purchased 10,000 copies of this leaflet to be donated to individuals and organizations in the same manner as their purchase of "Make Way for Even Teeth."

As of May 1, 1940, a total of 21,678 copies of "Facts" have been sold, and 2,679 given free.

Bulletins and Membership Participation.—Bulletin No. 1, "How to Use 'Make Way for Even Teeth,'" was mailed October 1, 1939, and the reception of the material was instantaneous as evidenced by orders for this reprint. The material accompanying the bulletin described the manner of distribution by purchasers. This bulletin went to the entire membership of the Association.

Bulletin No. 2, "Effective Public Speaking," was mailed February 15, 1940, also to the entire membership of the Association. This bulletin presented four typical radio talks on orthodontics, which had been selected from a number which had actually been delivered by members of the Association. Accompanying the bulletin were suggestions for the preparation of radio talks, gleaned from various expert sources.

It is important that in pioneer work in public relations efforts be made to inform the membership of the organization served, and allied organizations, of the progress of work. Items appeared as follows during the year:

American Journal of Orthodontics and Oral Surgery

June, 1939	Page 509	"Making the Truth Credible"
July, 1939	Page 671	"American Association of Orthodontists Establishes Public Relations Bureau"
September, 1939	Page 923	"Modern Methods for Orthodontic Lay Education"
October, 1939	Page 1022	"Public Relations Bureau, A. A. O. to Distribute Reprints"

December, 1939	Page 1234	"Public Relations—An Editorial"
February, 1940	Page 149	"Public Relations Bureau Prepares New Leaflet on Orthodontics"
April, 1940	Page 415	"Facts About Orthodontics for Health Workers"

Journal of the American Dental Association

February, 1940	Page 293	"American Association of Orthodontists Bureau of Public Relations"
April, 1940	Page 642	"Facts About Orthodontics for Health Workers"

The last document named above was a reprint entire of the leaflet of the same title published by the Bureau, reproduced with the consent of our Committee.

Regarding membership participation, it will be seen by reference to the orders for our material, that purchasers who are members of the society numbered 81 for "Make Way for Even Teeth" and 88 for "Facts About Orthodontics for Health Workers," to date. While at first glance this may appear to be a meager response from 660 members, if comparison be made with similar projects conducted on behalf of other organizations, it will be seen to be an encouraging response, especially for a pioneer effort.

Orthodontists are busy men. The time they can give to writing letters, sending orders, making contacts for the distribution of printed matter, is necessarily limited. They will devote time to such things only in proportion as they have knowledge of the working of the committee, and become sympathetic with its purposes, and then imbued with faith and zeal that will give them a sense of personal responsibility to help in their own towns and cities in the promotion of this educational effort.

The Director of the Bureau wishes to submit as his opinion that if each sectional society will set up a working, functioning committee, composed of men of initiative and executive ability who are willing to give time to swing into action on public relations projects, the American Association of Orthodontists will soon become a power commensurate with its skill and ability, in the dental and allied health educational fields. This is essential both to prepare the way for needed legislation and to preserve and maintain professional skills from the erosion of inadequately trained personnel. The potentialities of such a group are great, provided they can and will dispose sufficient time and energy in making themselves vocal and thereby become better understood and appreciated by the other professional health services, and the general public.

It is to be expected that a lag should exist between project initiation and its acceptance by the group. Another lag will occur between acceptance and kinetic activity. Experience in the medical field has amply demonstrated that the group on whose behalf public relations work is undertaken are the last to unitedly move into action to get the most out of their own activities. Outside groups are more quickly appreciative and cooperative, perhaps because work of this sort is ostensibly done for the benefit of these groups, though indeed it is a genuine obligation of any profession to educate the public and save them from the effects of ignorance and credulity. The fact should not be overlooked that in the end the result of increased acceptance of the group will be augmented esteem among persons whose opinion matters, which cannot fail to be translated into benefits of more practical significance, though it need not be emphasized nor sought as a direct end to be gained.

A considerable amount of correspondence with individual members of the Association who have expressed a specific interest in the activities of the Bureau has made the work of the Director a most enjoyable occupation. From unexpected sources have come communications which almost seemed to speak in person the genuine words of helpfulness which they contained. It has been a joy indeed to assist, during its crucial first year, a program which has elicited the interesting comments of these visitors in the morning's mail.

Contacts.—Correspondence has been maintained with many groups and persons outside the members of the Association. Among these are:

Fifteen State Departments of Health, seven City Boards of Education and sixteen Universities; the Ontario Dental Association, the Mexican Orthodontic Society, several dental

hygienist organizations, numerous Tuberculosis Associations, organized women's groups, and medical associations—state and local.

Our list of names of interested persons now numbers 475. They include persons who have ordered printed matter, or made inquiry in any way. It is suggested that such of these as are worth continued cultivation be placed in a file with a stencil made for each name and address. We now have the entire list of members of the Association on stencils, for economical addressing, and it will be desirable as our list is now rapidly increasing, soon to make stencils for the names of persons on a general "Cultivation List."

Future Projects.—It is proposed to publish a leaflet along popular lines entitled "Getting Ready to Tackle Life." Plans for this document are ready for presentation at the annual meeting in Chicago, May 13 to 16, 1940. An initial press run of 100,000 is suggested.

Continuation is planned of the distribution of "Facts About Orthodontics for Health Workers," to such lists as the American Public Health Association's 7,000 members, the American Board of Pediatric's 1,400 members, organizations of dental hygienists, Parent-Teacher Associations, and Federation of Women's Clubs. In order to get this type of document into the hands of persons who can best make use of it, the need will probably arise for a large free distribution. No organization can use a great number of this piece, which increases the distribution difficulty and cost. However, it is anticipated that the ease of distributing "Getting Ready to Tackle Life" will compensate to a great extent, so that probably the year 1940-1941 will show, on the whole, as favorable a financial statement as the past year.

If we can sell part of our publications to those able and willing to pay for them, the money can be put into free distribution to those unable to do so, achieving the greatest possible dissemination at the minimum of cost, which should be our aim.

If funds permit, a radio transcription might be a part of next year's public relations efforts. There will be on exhibition at the meeting at Chicago, an electrical transcription of the "Cavalcade of Dentistry" which will be produced there as an example of what can be accomplished by the use of this technique.

Repeated requests have come to the Bureau for an educational orthodontic exhibit to be used in the scientific exhibit at the A. D. A. convention at Cleveland, state dental societies, and other kindred health organizations. The need appears to be so urgent and desirable that a special appropriation should be made to finance such a project.

Financial Summary		*Expenditures*	
Appropriation	$2,500.00	Salaries	$1,450.00
Income from sale of literature	550.06	General and Misc.	1,367.70
Total	$3,050.06	Total	$2,817.70

Balance on hand, $232.36.

The committee recommends that this balance be reallocated to the Bureau.

Respectfully submitted,

FRANK NICOLAI
GEORGE A. BARKER
T. W. SORRELS, Chairman

Department of Oral Surgery and Pathology

Edited by

STERLING V. MEAD AND KURT H. THOMA

Articles on oral surgery, radiography, and anesthesia should be submitted to Dr. Sterling V. Mead, 1149 Sixteenth Street Northwest, Washington, D. C. Articles on oral pathology should be submitted to Dr. Kurt H. Thoma, 53 Bay State Road, Boston, Mass.

PATHOLOGIC FRACTURES OF THE MANDIBLE, PREDISPOSED BY OSTEOMYELITIS

REPORT OF A CASE

ARTHUR F. FISHER, D.D.S.*, AND W. R. J. WALLACE, D.D.S.†
ROCHESTER, N. Y.

NEARLY every bone in the body has been recorded as the site of a pathologic fracture. The weight-bearing long bones often have pathologic fractures, because of the great force exerted on them. In the mandible a break in the bone is not seen until the diseased area is well advanced and widespread.

Ghormley et al.,[1] Wilson,[2] Kolodny,[3] and others have given general classifications of pathologic fractures. Thoma[4] has listed the probable causes of pathologic fractures of the mandible as either local or systemic in character, the local ones being malignant tumors, benign tumors, infectious diseases, or cysts. The general diseases are osteogenesis imperfecta, hyperparathyroidism, Paget's disease, osteomalacia and rickets, atrophy, or marble bone disease.

There are very few case reports of pathologic fractures of the mandible in the literature. The largest number are found in bone lesions which are characterized by gross destruction with little or no osseous production. These generally fall into the group of local bone diseases. Hofer[5] has recorded twenty-five cases of spontaneous fracture of the mandible, all of which were local disturbances. Nine were from osteomyelitis, five from carcinoma, three from tuberculosis, and two each from tabes dorsalis, cystic tumors, giant-cell epulis, and sarcoma. Gurnick[6] reported a case of osteomyelitis arising from teeth extractions in the area. Cole[7] also reported a case following multiple extractions. In the American literature very few cases have been published. Field[8] reported a pathologic fracture of the mandible predisposed by osteomyelitis. The disease was a result of the removal of an impacted third molar. Field and Ackerman[9] also reported a case of fulminating osteomyelitis of the mandible with pathologic fracture. Again this diseased bone area was a result of teeth having been extracted.

From the Department of Dental Research and Surgery of The University of Rochester School of Medicine and Dentistry and Strong Memorial Hospital.

*Resident in Oral Surgery and Carnegie Clinical Fellow in Dentistry at the University of Rochester School of Medicine and Dentistry and Strong Memorial Hospital.

†Consultant in Dental Surgery, Strong Memorial Hospital.

Berger[10] claims that osteomyelitis and necrosis are the pathologic conditions which most often cause fracture and that these conditions may be caused directly or indirectly by infections about the teeth, syphilis, tuberculosis, and mercurial, arsenical, lead, or phosphorous poisoning.

Treatment.—The principles of treating pathologic jaw fractures are, if possible, to maintain proper relationship of fragments and preserve dental occlusion during and after treatment of the diseased bone. Continuity of the bone can be restored if necessary by bone grafts, but only after healing has occurred. Most writers advise conservative treatment wherever possible. Fixation should be of the simplest type. In malignant growths there is often severe pain experienced by the patient with almost any type of dental appliance. Hofer[5] points out that in these cases the patient's comfort is of primary importance.

Prevention.—Key and Conwell[11] advise that, if a growth is removed from the mandible and the danger of fracture is recognized due to the decreased volume of bone, interdental wiring is frequently resorted to for protection against possible fracture. Weinberg,[12] in dealing with pathologic fractures of any bone in the body, advises treating large bone cysts as simple fractures and immobilizing them at an early stage.

In view of the few cases of pathologic fracture of the mandible found in the literature, a case report is given below. It must be remembered that in spontaneous fractures the primary cause or pathologic condition is of great importance, while the immediate cause is usually of little consequence.[13] The patient seldom remembers the time or cause of the fracture. Trivial forces, such as rolling over in bed or opening the mouth, may result in the actual break in the bone.

Case Report.—Pathologic fracture predisposed by osteomyelitis. A woman (M. G.), 28 years old, was seen in the emergency room of Strong Memorial Hospital on Sept. 6, 1939. Ten days previous the patient was struck on the left side of the jaw by a fist. A few hours later the mandibular left second molar started "flashing pains." Pain, swelling, and trismus progressed despite the warm compresses she applied to her face. Because of home conditions, the patient would not stay in the hospital that night, but the following day she was admitted.

Physical examination showed marked trismus, swelling and tenderness of the left ramus of the mandible, and no obvious bone defect. The mandibular left second molar was painful to percussion although firm in the socket. The x-ray was negative (Fig. 1).

Heat was applied to the left side of the face with no effect. On Sept. 17 the patient's condition was unchanged. Incision and drainage of left jaw were carried out under gas, oxygen, and ether. (All external incisions were done by Forrest Young, M.D.) No pus or abscess pocket was found. On Sept. 26 the patient's condition was unchanged and a second similar operation was carried out. This time about 5 c.c. of pus were removed from the submaxillary triangle region.

An x-ray taken Oct. 2 (Fig. 2) showed a pathologic fracture of the left jaw in the second molar region and localized osteomyelitis in that area. The mandible was immediately put to rest with a chin support.

X-ray on Oct. 9 (Fig. 3) showed increased osteomyelitis about the mandibular left second molar area with sequestration of the middle portion.

On Oct. 24, under gas, oxygen, and ether, a sequestrum was removed, and the tooth in the line of fracture was gently extracted. The pathologic report of the sequestrum suggested chronic osteomyelitis. The following day the teeth

Fig. 1.

Fig. 2.

Fig. 1.—X-ray of patient taken Sept. 7, 1939.
Fig. 2.—X-ray of patient taken Oct. 2, 1939.

were wired together with simple vertical wiring (Ivy's eyelet method). Pus drained from the external incision for ten days. An x-ray taken Nov. 1 (Fig. 4) showed the space being filled in with bone. On Nov. 10 the patient was discharged from the hospital with the wires still in position.

The patient returned to emergency on Nov. 16 and said she had been struck on the right side of the face the day after her hospital discharge. Swelling started three days later on the left side. X-ray taken Nov. 18 showed little change (Fig. 5). Patient was again admitted to the hospital because of the considerable amount of external drainage. The treatment consisted in keeping the patient quiet in bed and gradually the swelling and discharge decreased.

Fig. 3.

Fig. 4.

Fig. 3.—X-ray of patient taken Oct. 9, 1939.
Fig. 4.—X-ray of patient taken Nov. 1, 1939.

On Nov. 28 the wires were removed, and the patient was discharged from the hospital.

The patient was followed in the outpatient department and an x-ray, taken Feb. 21 (Fig. 6), showed good bony union at the original fracture site.

Fig. 5.

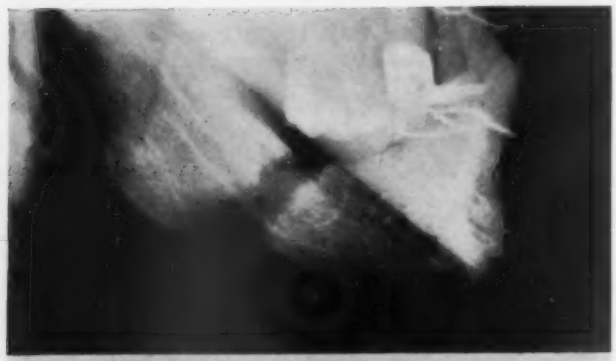

Fig. 6.

Fig. 5.—X-ray of patient taken Nov. 18, 1939.
Fig. 6.—X-ray of patient taken Feb. 21, 1940.

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ORAL MANIFESTATIONS OF LEUCEMIA

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FOR a better understanding of the etiology of oral manifestations of leucemia, 190 cases seen at the Cleveland Clinic during the last five years were reviewed. The oral condition may be due to (1) the loss of the protective elements in the blood, (2) the loss of the clotting factors in the blood, or (3) the infiltration of the oral mucosa by the leucemic cells. The oral signs may be entirely lacking, or when present, they may be most severe.

Haden[1] defines leucemia as an abnormal, unrestrained, overgrowth of leucopoietic tissue. He further classified three leucemic phases as: (1) the leucopenic phase, showing a reduction of the total white count; (2) the normal white count; and (3) leucocytosis phase, showing an increase in the total white count. The increased number of leucocytes with a reduction of red blood cells and color index in the circulating blood reflects the overactivity of the tissue-forming white cells.

Leucemia may occur in either the acute or chronic stage. Three chief varieties are: myelogenous leucemia, characterized by an increase in myelocytes and myeloblasts in the blood stream; lymphatic leucemia, by an increase in lymphoblasts or lymphocytes; and monocytic leucemia or reticulo-endotheliosis, by an overproduction of monocytes in the blood stream. Difficulty in diagnosis of the type of leucemia often arises from improper identification of the leucocytes, particularly in the early stages of development.

Haden,[1] Forkner,[2] and others described ulceration and necrosis of the gingival tissues as pathognomonic of the monocytic variety. Haden stated that gangrenous lesions of the mucous membrane characterize chronic monocytic leucemia. In acute monocytic leucemia, Forkner found the gingiva to be diffusely swollen with the teeth tending to become submerged in the gum. A diffuse cellulitis may be present causing pain and signs of an acute inflammation extending into the deeper tissues of the face. On biopsy, the gingival tissues of one of his patients showed a diffuse infiltration of leucemic cells. He also stated that bleeding from the gums is common in all types of acute leucemia.

Favreul and Landais[3] reported noma in patients with acute leucemia. Prinz and Greenbaum[4] described the infiltration of the dental alveolar periosteum with subsequent loosening of the teeth. Comroe, et al.,[5] described the gingiva as red, spongy, and swollen with other mucous membranes contrastingly pale. He stated that the ulcerative lesions may involve any portion of the mucosa and may progress to extreme necrosis with severe pain and fetor oris.

In the 190 cases of leucemia reviewed, eighteen were incomplete regarding oral manifestation. Of the remaining 172 cases, thirty or approximately 18 per cent exhibited some form of oral manifestation.

From the Cleveland Clinic.

In twenty-seven cases of monocytic leucemia, thirteen or approximately 50 per cent had oral manifestation. Of twenty-one cases in the acute stage, eleven or 52 per cent showed oral manifestations, nine of which were in the leucocytosis phase. Ulceration and necrosis of the oral mucosa were present in eight. In one edentulous patient who wore dentures, a small lesion developed on the hard palate, increased rapidly in size, and became necrotic in the center. In five patients, the onset of the oral condition followed dental extraction. Hypertrophy of the gingival tissues was characteristic except in the edentulous case cited above.

In many cases of monocytic leucemia without oral lesions, the patients were edentulous, or the diagnosis was not clearly established. However, two cases of acute monocytic leucemia in the leucocytosis phase, with apparent oral neglect, had no oral manifestations. One patient with a final diagnosis of chronic monocytic leucemia previously presented a picture of thrombocytopenic purpura when extensive bleeding followed extraction. Later, under hospitalization, other teeth were removed with surgical preparation of the alveolar bone, without postoperative complications. The following three cases are representative of this group.

Case 1.—A 40-year-old man who had been in good health up to his present illness gave the following history: Two weeks previously, his dentist had referred him to his physician because of an abnormal gum condition. Blood study revealed a mild anemia and a marked leucopenia, with concentrated preparations showing many monocytes, some quite immature. The spleen and glands were not enlarged. The patient developed some petechiae, subcutaneous hemorrhages, and a definite necrosis of the gum margin around a number of the teeth. The gingiva bled slightly and fever was present every day. Weakness was the only other symptom. The patient declined in spite of transfusion and died one month after the first oral examination by the dentist.

Case 2.—A 45-year-old man had been in good health until eight weeks prior to the present examination. Following extraction of five posterior teeth because of pyorrhea, he had a continuous hemorrhage for eleven to twelve hours until finally controlled by his physician. However, the patient bled intermittently and became paler and weaker. He was hospitalized for three weeks without improvement. Shortly after extraction, the patient noticed the progressive enlargement of the cervical glands, which were treated by roentgen therapy. Petechiae on the lower extremities were noted one month prior to examination. There was marked fetor oris. The superior surface of the tongue and the gingiva on the left side of the mouth were edematous and covered with a green, brownish, indurated blood clot. The blood study revealed a marked anemia, a normal white count with 55 per cent monocytes and numerous monoblasts, and very few platelets. (The follow-up history of this patient was incomplete.)

Case 3.—A 41-year-old housewife, whose chief complaint was the condition of her mouth, gave the following history: One and a half months previously, she had had a tooth removed. Two days later she developed a condition diagnosed as "trench mouth" which necessitated hospitalization. At the time of the examination at the Clinic, the patient had a marginal gingivitis with hyper-

trophy of the gingiva, almost covering the teeth, which were loose and tender to percussion. The submaxillary glands were enlarged. Smears from the gingiva were strongly positive for fusospirochetal organisms. There was fetor oris. The blood study revealed a marked anemia, 16,000 white cells, of which 57 per cent were monocytes, and normal blood platelets. A diagnosis of monocytic leucemia was made. By local treatment to the mouth, the gingival condition was improved slightly, and the patient was made more comfortable. Gradually, however, a large necrotic area developed on the lingual surface of the mandibular left molar region with considerable induration of the soft tissues, and pain. Roentgen examination revealed a sclerosing type of osteomyelitis. The patient declined rapidly and died four months after the initial examination.

Case 4.—A 40-year-old man was admitted to the hospital in 1936 with a complaint of bleeding from the gums for fifteen days posteroperatively. Three weeks previously he noticed a slight tenderness in the maxillary right gingivolabial region which his dentist diagnosed as a "gum boil." The boil was incised but contained only blood which continued to flow intermittently. The patient always considered himself to be in good health, but had lost sixteen pounds in two years in spite of a good appetite. He gave a history of hemorrhage following tonsillectomy with six weeks' hospitalization fifteen years previously. Physical examination (1936) revealed a blood pressure of 140 mm. systolic and 100 mm. diastolic. Petechiae appeared on tourniquet testing. The mucous membranes were of good color. Bleeding behind a soft clot on the labial surface of the upper gingiva was observed. Blood examination revealed 5,300,000 red cells, 81 per cent hemoglobin, 12,450 white cells with 89 per cent neutrophils, 7 per cent leucocytes, 4 per cent monocytes, and 35,000 platelets. The coagulation time was twenty minutes and bleeding time, fifteen minutes. A diagnosis of toxic thrombopenic purpura was made. In 1937 the platelet count rose to as high as 110,000. The blood count in December, 1937, was as follows: 16,000 white cells, with 65 per cent neutrophils, 80 per cent lymphocytes, and 25 per cent monocytes.

In 1938, the patient was readmitted to the hospital because of an upper respiratory infection with nasal congestion. During the interim, he had been in and out of the hospital several times. He felt better but continued to be very weak and to bleed slightly from the nose and gums daily. The blood count in January, 1939, was: 3,460,000 red cells, 45 per cent hemoglobin, and 39,250 white cells, of which 49 per cent were neutrophils, 5 per cent lymphocytes, 12 per cent monocytes, and 30,000 platelets. On the last hospital admission, the patient steadily lost ground in spite of blood transfusion. Bleeding increased from the mouth, and the mucosa was markedly anemic. A large clot gathered in the left cheek near the corner of the mouth, and gradually became necrotic, with the adjacent tissues becoming indurated. The diagnosis was monocytic leucemia with marked oral involvement simulating noma formation. The patient died thirty-three days following the last admission.

In this series of 172 cases, seventy-seven were of lymphatic leucemia, nineteen being in the acute stage. Of the seventy-seven cases, six or less than 1 per cent had oral manifestations. Three or approximately 16 per cent of the cases with oral manifestations were in the acute stage and three, or 15 per cent,

in the chronic stage of the leucocytosis phase. Two patients with a diagnosis of lymphatic leucemia developed gangrenous lesions of the hard palate and tonsillar regions. One, an 8-year-old boy in the leucopenic stage of leucemia with 1,000 white cells and 92 per cent lymphocytes, had a marked anemia and no platelets. In addition to the gangrenous lesions of the hard palate, he had hypertrophy of the gingival tissues with a bleeding diathesis. The second patient, a 67-year-old man, had a leucocytosis with 78 per cent lymphocytes, anemia, and a marked reduction of blood platelets. He exhibited extensive oral neglect, gangrenous lesions of the tonsil, and enlargement of the lymphatic glands of the neck. One case of chronic lymphatic leucemia in a leucocytosis phase was terminated by an extensive loosening of the teeth, in which the dentist picked out several and several more were expectorated by the patient. Stomatitis was extensive with a slough extending over the floor of the mouth, one side of the tongue, gingivae, and the hard palate. The oral cavity had previously exhibited gross oral neglect.

In the remaining seventy-one cases of lymphatic leucemia without oral manifestations, approximately twenty-five per cent had oral conditions described as gross neglect. Many of these patients were partially edentulous, and approximately 29 per cent were completely edentulous. In most of the cases, the reduced blood platelet count seemed to have no effect upon the oral condition.

One patient, a 61-year-old man, with extensive pyorrhea and a primary complaint of an eye condition, had a complete prophylactic odontexesis and gingival medication prior to the removal of three teeth. Following the removal of the teeth, the blood report classified him as having an acute lymphatic leucemia in a leucocytosis phase. Slightly palpable glands also were discovered. In spite of the blood findings, including reduced blood platelet count, no complications followed extraction, with a well-organized blood clot and normal healing of the sockets. In another patient who had no other oral manifestations but oral neglect, and a diagnosis of chronic lymphatic leucemia in the leucocytic stage, two teeth were removed with no apparent complications two months prior to his visit to the clinic.

The following case is an example of the oral manifestations of lymphatic leucemia. The patient was a 30-year-old colored housemaid who came to the clinic with a complaint of sore gums. One month previously, she had two wisdom teeth removed. A few days later, her lower gums began to pain and burn with some generalized swelling. She found it increasingly difficult to eat. During the month prior to the consultation, she continued to work, in spite of feeling ill with frequent headaches. During this time, she was treated by her dentist for fusospirochetal infection.

Examination revealed a colored woman of 155 pounds with a blood pressure of 120 mm. systolic and 70 mm. diastolic. The oral examination revealed a coated tongue and dusky red-colored hypertrophied gums, which were very tender and bled fairly easily. Several tender lymph nodes were observed under the mandible. The blood study revealed 2,600,000 red cells, 31 per cent hemoglobin, and 18,000 white cells, of which 82 per cent were lymphocytes. A diagnosis of acute lymphatic leucemia was made. The patient died one week following this consultation.

In this series of 172 cases of leucemia, sixty-eight cases were diagnosed as acute or chronic myelogenous leucemia, eleven or 15 per cent of which had oral manifestations. In twenty-six cases of acute myelogenous leucemia, 1.9 per cent had oral manifestations, as comparable to 1.4 per cent in the chronic type. There was an equal incidence of oral manifestations in the acute stage of both the leucopenic and leucocytosis phases. In the chronic stage, the greatest incidence of oral involvement was in the leucocytosis phase.

Many of fifty-seven cases of myelogenous leucemia without oral manifestations showed reduction in the blood platelets and oral neglect. One patient with a blood dyscrasia complicated by diabetes, arteriosclerosis, and oral neglect, had no oral symptoms. During the course of the disease, one patient had all the maxillary teeth removed without complications. Two patients without oral symptoms dated the onset of the blood dyscrasia to the time of dental extraction, one with a history of a dry socket, and the other with gingival bleeding. The oral manifestations in myelogenous leucemia were characterized by bleeding gingivae. Gingival hypertrophy or mucous membrane ulceration was not present in spite of apparent oral neglect. The following cases are characteristic of the group with oral manifestations.

Fig. 1.—Colored patient with acute lymphatic leucemia seen in the Dental Department of the Cleveland Clinic for Vincent's infection after three months of treatment. The patient died shortly after consultation. The patient showed gingival hypertrophy.

Case 1.—The patient was a 29-year-old housewife who had been well until six months previous to the present examination. At that time, she noticed bleeding from the gums, a shortness of breath, palpitation, and generalized weakness. However, she continued to do her housework for three months before consulting her own physician. During treatment, a vision defect developed, and she was referred to the clinic for consultation.

Physical examination revealed a fairly well-developed young woman weighing 140 pounds with a blood pressure of 110 mm. systolic and 60 mm. diastolic.

The skin was pale, smooth, and dry with a few petechiae. Many of the teeth were crowned. The gingiva appeared slightly edematous with an oozing hemorrhage. The tonsils were injected. Examination of the eyes revealed a hemorrhagic retinitis. The blood study revealed 1,700,000 red cells with 33 per cent hemoglobin, and 3,200 white cells with 18 per cent abnormal forms of myelocytes and myeloblasts; the platelet count was 40,000. The bleeding time was sixteen minutes. A diagnosis of acute myeloblastic leucemia was made. The patient declined rapidly while confined to the hospital for diagnostic study. The final outcome was not recorded.

CASE 2.—A 46-year-old housewife was first seen relative to a complaint of a very large spleen. She said that the discomfort began in the abdomen five years previously, when she had been confined to her bed for a period of seven weeks with severe sweating and weakness. The blood study at that time revealed a low red cell and white cell count. Physical examination at the clinic revealed a very large spleen. The teeth were in very poor repair. The blood examination showed a marked leucocytosis with a high percentage of myelocytes. A diagnosis of chronic myelogenous leucemia was made. Under treatment including roentgen therapy to the spleen, the patient improved and was discharged after a period of two weeks' hospitalization. Seven months after discharge from the hospital, she was readmitted because of extensive and continuous bleeding from two sockets following extraction two weeks previously. The patient was very weak and apprehensive, with a very black mouth, considerable fetor oris, and a coated tongue. There was no bleeding from the nose or actual petechiae of the skin with the arm cup (Rumple-Leeds phenomena). The white cell count had greatly increased. The blood platelets at the time of the initial examination were abundant, and follow-up blood examinations revealed a long period of leucopenia. Under transfusion treatment, the patient at first showed a slight improvement with decreased oral bleeding. However, in spite of treatment,

TABLE 1

	ACUTE MONOCYTIC LEUCEMIA			CHRONIC MONOCYTIC LEUCEMIA			ACUTE LYMPHATIC LEUCEMIA			CHRONIC LYMPHATIC LEUCEMIA			CHRONIC MYELOGENOUS LEUCEMIA			ACUTE MYELOGENOUS LEUCEMIA		
	LEUCOPENIA	NORMAL W. B. C.	LEUCOCYTOSIS	LEUCOPENIA	NORMAL W. B. C.	LEUCOCYTOSIS	LEUCOPENIA	NORMAL W. B. C.	LEUCOCYTOSIS	LEUCOPENIA	NORMAL W. B. C.	LEUCOCYTOSIS	LEUCOPENIA	NORMAL W. B. C.	LEUCOCYTOSIS	LEUCOPENIA	NORMAL W. B. C.	LEUCOCYTOSIS
Positive oral manifestation	1	1	9	1	0	1	1	1	1	0	0	3	2	1	2	1	2	3
Negative oral manifestation	1	1	8	2	2	0	5	0	11	4	4	47	6	4	11	4	3	29
Total cases	2	2	17	3	2	1	6	1	12	4	4	50	8	5	13	5	5	32

NOTE: In 190 cases reviewed, eighteen were inconclusive relative to oral findings and were not included with the above findings.

the patient continued to decline, gingival bleeding continued, and generalized petechiae developed over the body. Within two months following the dental extraction, the patient died.

The etiology of the oral manifestations of leucemia has not been established. Eighteen per cent of 172 cases of verified leucemia showed oral manifestations. Bleeding diathesis appeared in all types of leucemia. Hypertrophy of the gingiva was more frequent in the lymphatic and monocytic types. In monocytic leucemia, the condition may progress to necrosis and simulate noma in some instances which may be regarded as a differential sign of this type of leucemia. The frequency of oral neglect and decreased platelet count does not entirely explain the oral manifestations.

Oral cleanliness may influence the oral symptoms. Hypertrophy and excessive vascularity may be a result of some cellular infiltration, and sloughing to extensive necrosis may depend upon invading bacteria on the weakened changed oral structures of the extremely debilitated patient. The frequent finding of alveolar resorption also suggests the infiltration of the alveolar bone with hematopoietic tissue and marked osteoclastic reaction. Frequently, the earliest manifestations of this blood dyscrasia may be noted in the mouth. The oral surgeon should be ever alert to the possibilities of other factors not of local origin in evaluating and diagnosing oral disease.

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HYPERTROPHY OF THE GUMS DUE TO THE ADMINISTRATION OF DILANTIN SODIUM

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DILANTIN sodium was introduced to the medical profession to be used in the treatment of epilepsy during the 1938 meeting of the American Medical Association held in San Francisco. This drug is a sedative and one of the barbiturate group. There have been at least twenty-nine clinical reports published during this brief time in the medical literature, but none could be located in our dental literature as to its effects on the gum tissue.

The following case report is one that may be of interest, should one of these cases be contacted.

This case report is of a young white man, 17 years of age, an American. On examination of the oral cavity an advanced hypertrophy of the gum tissue was noted. There was an excessive overbite of the anterior teeth with slight crowding of the mandibular incisors.

The hypertrophied tissue was very firm in character, with no discharge, odor, or bleeding from trauma nor any signs of disintegrating margins. The roentgenogram showed no apparent bone change from normal. There was no tendency toward decay.

His general clinical history was found to be as follows:

Since the age of 14 he had suffered from epileptic convulsions of the grand mal type.

In June, 1939, treatment with dilantin sodium was started and although he had averaged seven seizures a week for the previous two years, he did not have a seizure in nine months in which he received the drug.

Ten days after commencing treatment he complained of soreness of the gums; this disappeared in about two weeks. In about two months enlargement of the gums was evident to the patient, and to his family. In another month it had progressed to such an extent that his dentist advised surgical removal of some of the tissue. This was not undertaken.

A second dentist was consulted six weeks later who took a smear and pronounced the condition to be a case of Vincent's infection.

The parents not being satisfied with this diagnosis sought the assistance of an oral surgeon to have some of the gum tissue removed. The oral surgeon, not being able to classify the condition, hesitated to operate, and referred the case to me, as he felt trauma might be responsible for the condition of the tissues.

The patient's general health was good, mental condition and general attitude had greatly improved. Mouth hygiene was excellent.

The etiology of the hypertrophy was felt to be due to a deficiency of vitamin C, the system having been robbed of this substance by the administration

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of the dilantin sodium. No dental therapy was undertaken. The patient has moved to another state and no further information will be available on his case.

In a report by Kimball[1] he states that 57 per cent of the patients treated by him more than two months with the drug showed signs of gum changes. It appears that the higher the dose of the drug, the more advanced the hypertrophy. The ascorbic acid content of the blood also showed a decrease in proportion to the increase in hypertrophy. The other symptoms usually found in prolonged and severe cases of vitamin C deficiency have not been noted.

However, studies made by Parke, Davis and Co.[2] on the vitamin C content of the blood of rats receiving large doses of dilantin sodium showed no decrease in this vitamin.

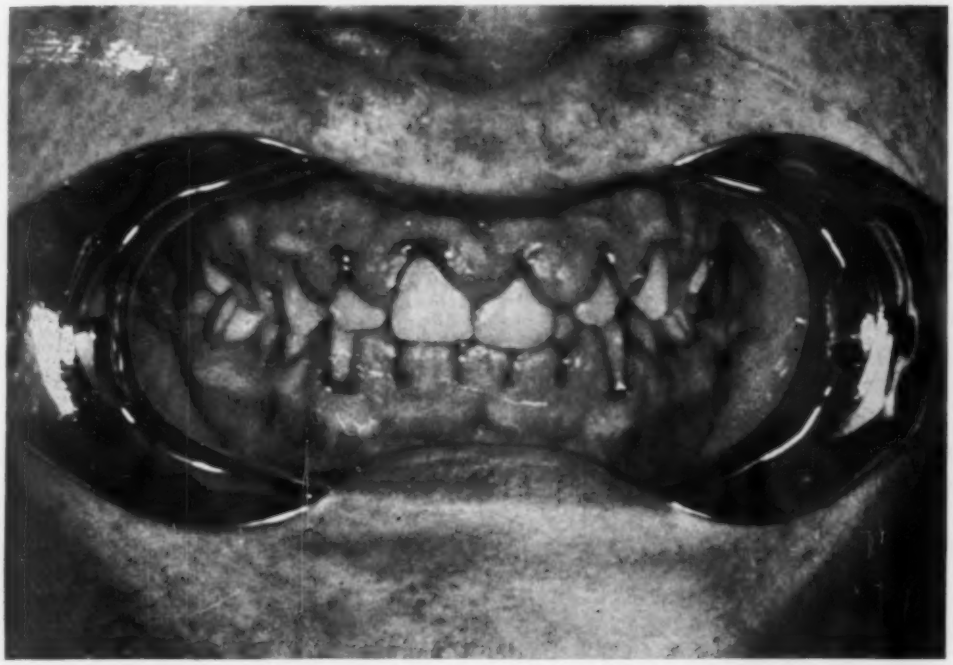

Fig. 1.—The extent of the hypertrophied gum tissue is shown.

All of the reports made to date have been preliminary reports and no doubt the final reports will give more information as to the true dental condition.

After the proper dosage of dilantin sodium alone or in combination with other sedatives has been worked out, less toxic effects can be expected. It would seem that if vitamin C is a factor, it would also be wise to administer large enough amounts from the outset of treatment to keep a normal balance. This would keep the possibilities of gum hypertrophy down to a minimum.

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1158 Roosevelt Bldg.

DOUBLE ELONGATIONS OF PARTIALLY CLEFT PALATES AND ELONGATIONS OF PALATES WITH COMPLETE CLEFTS

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A METHOD of elongating partially cleft palates was described in 1936,* the principle of which is that practically the entire soft tissue of the palate is freed from the bone, the major palatine arteries are loosened but not cut, and the entire mass of tissue is immediately set back—as a direct flap with preservation of its arterial supply—so that the anterior part, from just behind the incisors, is anchored all the way back at the posterior border of the bone. A gain in length is thus obtained equal to about as much as the expanse of denuded bone, the object being to have the longest possible functioning palate, to assist in nasopharyngeal closure in speaking and eating.

Some of the original illustrations of the procedure in Figs. 1 to 4 show the complete freeing of the tissues from the bone, the preservation of the arteries and the anchoring of the palate to a little bridge of nasal mucosa that has been left behind for this purpose. The closure of the cleft itself is done usually at the same operation—as a preliminary step—in children, but may be done as a separate operation. In adults two stages are advisable because of excessive bleeding. The sequence of the elongation and the closure does not have to follow a definite rule; therefore, a field is opened for older children and even adults, who have had clefts closed many years before, to have their palates elongated in an effort to obtain better speech.

Observations made or emphasized since the original publication may be summarized, as follows:

1. It is still thought that the direct flap, elevated and immediately set back, so that the least possible thickening will take place, is superior to the delayed flap method of elongation.

2. Preservation of the major palatine arteries (often called the posterior) is possible in nearly all patients, and freeing is effected by careful loosening of all tissue around the artery, gently stretching it from the foramen and, if necessary, carefully cutting it away from the palate flap. These methods have seemed better than trying to dislodge the artery from its bony canal by trying to remove the posterior wall of the canal.

The palate may be gotten so free by this dissection plus complete separation of the aponeurosis from the bone that it may be easily "set-back," practically against the posterior pharyngeal wall in most instances. This finding is in contradistinction to that of others who think that preservation of the arteries prevents elongation.

This may seem somewhat of an equivocal point but it makes possible closure and elongation in a single stage, and it may give a less scarred and

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more pliable palate. If the arteries are cut, then the delayed flap method of Dorrance may be resorted to for safety's sake. However, if they have been lost at a *previous* operation, then the delayed advantage has already been gained and the flap could probably be used without further delay.

3. This method of elongation, even though involving a very wide dissection, does not seem to interfere with the levators in the sphincteric action that attempts to close the nasopharynx. Even as the patient is waking in the operating room, very marked sphincter action can be seen.

4. In any procedure in which the posterior pillars are cut low down in the pharynx and then sutured behind the uvula to lengthen the palate, a tight scar band may be left right in the region of the greatest action; and if there is any

Fig. 1.

Fig. 2.

Fig. 3.

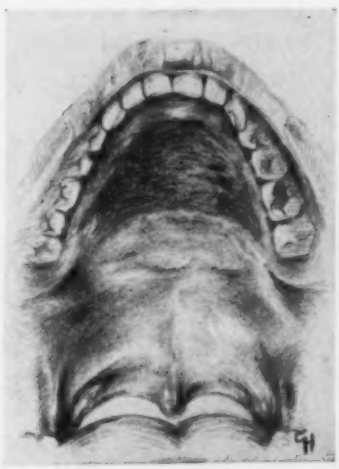


Fig. 4.

Fig. 1.—Palate detached from bone, but with both major palatine arteries intact. A little triangle of nasal mucosa has been left behind to which the palate is attached with the first suture. (All figures are diagrammatic.)

Fig. 2.—Detail diagram of deep separation of the soft tissues around the artery; exposure and fracture of the hamulus and complete separation of the aponeurosis from the bone.

Fig. 3.—Completion of the elongation. The palate has been set back and anchored as shown. Several layers of iodoform gauze soaked in balsam of Peru are placed smoothly and firmly over the exposed bone and may be removed in 5 to 7 days. The closure of the cleft may be done at this time, at a subsequent step, or may have been done at an earlier operation.

Fig. 4.—Completed elongation and closure after healing has occurred, with about as much gain in length as the expanse of original bone in Fig. 3.

levator action present, this procedure should be omitted if possible. The same is true of the employment of flaps from the posterior pharyngeal wall attached to the palate. These flaps may nearly or completely occlude the nasopharynx and also may give a "drum-head" noise from vibration in breathing.

5. If there has been a slough of the soft palate from previous operations, then, added to the elongation, may be the freeing and suturing of any tissue available, plus the use of a posterior pharyngeal flap attached to the palate if desired. This results in all possible closure between the mouth and nose, and, as there is no levator action, amounts to a flesh obturator. This, of course, has a great advantage over a dental prosthesis or over an extraoral flap.

6. Any type of pharyngoplasty, mainly that of Wardhill, in which there is a reefing forward of the superior constrictor may be added to this elongation procedure, but it has not proved as successful in my hands as in others'.

7. Tooth buds may be damaged as in any other flap elevating operation, if done too early; therefore, the elongation can be delayed until the second year or later if desired.

8. Results of the closure and elongation have been increasingly hopeful. The bone covers over in about 30 days; the palates usually smooth out, appear good and long, and, for these reasons, the contracture on the nasal surface in healing does not seem great enough to warrant trying to skin graft the raw surface.

Speech improvement has been almost dramatic in many instances, and the use of voice recording, either by simple direct methods, or electrical transcription has been of great help in permitting the patient to hear his own defects and thereby try to correct them. Speech training is important in all patients and the author is pleased to acknowledge invaluable help from Dr. M. A. Goldstein, Miss Mildred McGinnis, and other teachers of speech at the Central Institute for the Deaf and in the St. Louis public school system, and to Mr. C. E. Harrison of the Technisonic Laboratories, located in St. Louis, for his work with electrical transcriptions.

Most patients are also able to eat better than before the elongation.

9. Failures may be expected as more difficult palates are operated upon, that is, ones badly scarred and with tissue lost from previous attempts. Patients with as many as 18, 24, 45, and 55 operations to their credit in attempted repairs of lip and palate clefts have been seen.

10. Throughout the procedures of elongation there has been the fundamental aim that no opening should be left anteriorly, to avoid the use of an artificial dental obturator for closure of the mouth from the nose.

11. Acknowledgment is made to Dorrance for his pioneer work in calling attention to the advantage of the elongated palate and also for coining the term, "congenital insufficiency of the palate," for those instances of short palate with cleft palate speech but without an actual cleft.

12. Other methods of incision which leave a "V" of tissue over the bone, but necessitate more closure down the midline may have the immediate result of a smoother surface but give the added possibility of holes through the thin tissue that is sutured.

DOUBLE ELONGATIONS OF PARTIALLY CLEFT PALATES

In some partial clefts of the palates there is only a narrow ledge of bone just inside the alveolus with corresponding lack of mucoperiosteum with which a closure might be effected. In some of these the cleft may be so wide that even simple closure may be questionable.

Author's Technique.—In these patients it may be possible to do a double elongation of the palate, by first setting the available tissue back to the posterior border of the bone and then, after there is firm healing, further lengthening the palate by splitting the originally elongated part in two, from the edge of the bone backward, and from side to side. Care should be used not to open through the nasal mucosa, and the anterior free edge of the palate should be anchored entirely back on its own nasal mucosa (Figs. 5 to 8).

Fig. 5.

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 5.—Diagram of a cleft with only a narrow band of bone and soft tissue, showing incision for first stage of a "double set-back."

Fig. 6.—Healing resulting from the first stage set-back and with incision for the second stage of the "double set-back."

Fig. 7.—Completed double set-back, accomplished by carefully splitting the remaining palate tissue in two from before backward and from side to side without breaking through into the nose—and literally setting the palate back on itself. The arteries usually have to be sacrificed in this double procedure. This gives a double gain in length without an opening into the nose anteriorly. The raw surface closes over in 2 to 4 weeks.

Fig. 8.—Completed double set-back and closure of the cleft with about as much gain in length as the distance from the palate edge to the incisors and without any opening into the nose.

The closure of the cleft can be done at any one of the stages or as a separate procedure. The denuded nasal mucosa and fibrous tissue heals over as readily as the denuded bone does and, on late observation of the roof of the mouth, some trouble may be had in even discerning that the areas are healed with "scar epithelium." The steps of the procedure are most easily followed in the illustrations and their legends (Figs. 5 to 8). The arteries will have to be sacrificed at the second stage, but it is apparently not necessary to revert to a delay in setting the flap back, unless a hole has been made through into the nose.

ELONGATIONS OF PALATES WITH COMPLETE CLEFTS

As a corollary to the finding that the nasal mucosa could be saved in double elongations and in palates with submucous bony clefts, it has been

Fig. 9.

Fig. 10.

Fig. 11.

Fig. 9.—A complete cleft of the palate that has been closed successfully in the usual lateral flap manner, showing incision for elevating the palate.

Fig. 10.—Palate freed from the bone and from the nasal mucosa by carefully splitting the palate in two from side to side and from before backward and elevating it from the nasal mucosa without breaking through into the nose. About one-half of the thickness of the palate is left along with the nasal mucosa.

Fig. 11.—An elongation or set-back of a complete cleft of the palate finished, with the anterior edge of the palate all the way back at the posterior bony edge and anchored to the nasal mucosa. No opening exists into the nose and the raw surface will cover in 2 to 4 weeks. It is possible that the arteries may have to be sacrificed in carrying out this dissection, but the flap does not have to be delayed, unless there has been an accidental opening into the nose.

determined that elongations may be done in complete clefts of the palate that have been successfully closed at a preliminary operation. This elongation is also done to avoid an anterior opening into the nose and, therefore, to avoid the need of an artificial dental obturator.

Author's Technique.—The elongation is carried out as described for the partial clefts except that extreme care is taken to split the tissue in two from side to side and from before backward, leaving about one-half the thickness of the palate and, of course, the nasal mucosa behind. If even a small opening were made through into the nose, it would be best to replace the flap and elevate it later, with another attempt at not opening into the nose. The arteries may have to be sacrificed and, if there is any question of the blood supply, a delay in setting the palate back should be made. However, it is usually possible to go ahead with the elongation in a single stage (Figs. 9 to 11).

More difficulties in general can be expected in this procedure, and observations are limited for the present, but it makes possible a definite hope for improvement in speech in many patients who have had complete clefts simply closed many years before.

Case Reports

Case reports for this section of the Journal should be sent to Dr. Kurt H. Thoma, 53 Bay State Road, Boston, Mass.

CASE REPORT NO. 42

THIRTEEN IMPACTED AND UNERUPTED TEETH IN ONE MOUTH

P. Philip Gross, D.D.S., Philadelphia, Pa.

THE patient was a white man, single, 38 years old, with no family. He presented himself for dental examination with no specific complaint.

The intraoral examination revealed only nineteen teeth fully erupted. The patient claimed that he had had none extracted.

Roentgen examination revealed the following impacted and unerupted teeth (Fig. 1):

Mandibular right third molar
Mandibular right second molar
Mandibular right second premolar
Mandibular left second premolar
Mandibular left third molar
Maxillary right third molar
Maxillary right second molar
Maxillary right second premolar
Maxillary left canine
Maxillary left first premolar
Maxillary left first molar
Maxillary left second molar
Maxillary left third molar

Comment.—The patient stated that he had no pain or discomfort at the present and would not consent to surgery.

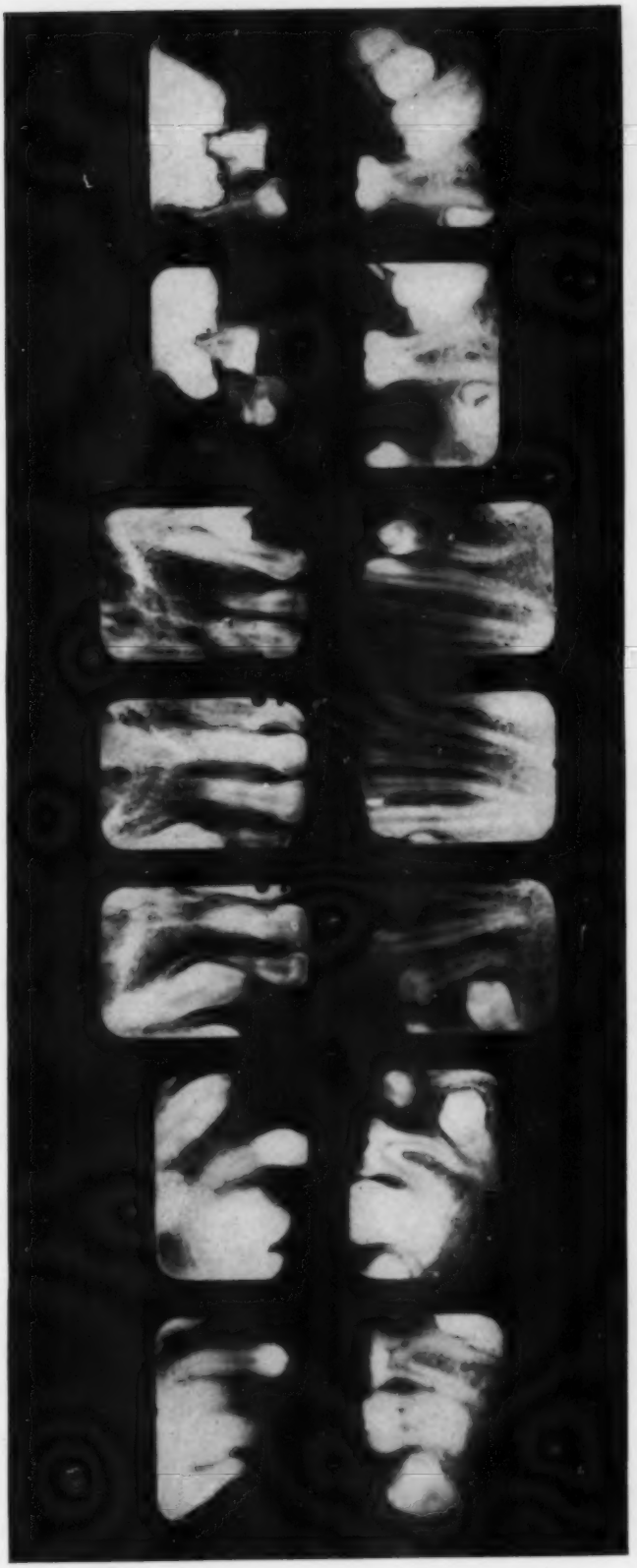


Fig. 1.

Department of Orthodontic Abstracts and Reviews

Edited by

Dr. J. A. Salzmann, New York City

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City.

The Enamel of Human Teeth: By M. Diamond, D.D.S., and J. Weinmann, M.D., *Columbia Dent. Rev.* **11**: 7, May, 1940.

These authors differ with the presently accepted theory on enamel calcification which is to the effect that the impregnation of calcium salts into the soft organic enamel matrix formed by the ameloblasts starts at the dentino-enamel junction and proceeds toward the periphery of the crown in successive incremental layers of organic enamel matrix each in turn impregnated by calcium.

According to Diamond and Weinmann, the entire organic enamel matrix is formed at once and contains the hardened calcium salts in a colloidal state. The crystallization of the colloidal calcium does not take place in the direction of the dentino-enamel junction but begins, on the contrary, at the highest peripheral point on the external contour of the crown and then radiates slowly in the direction of the dentino-enamel junction, or in a transverse direction to the incremental lines of growth.

Fig. 1.—The entire adult thickness of enamel in a state of organic matrix is seen at *A*. *B*., a characteristic artifact due to shrinkage between the dentin and enamel matrix, and erroneously interpreted as evidence of fully matured enamel. *C*, Reduced enamel epithelium. *D*, Odontoblasts. *E*, Pulp.

This new fundamental knowledge is important to the better understanding of the problems regarding enamel in relation to deficiency lesions. These find-

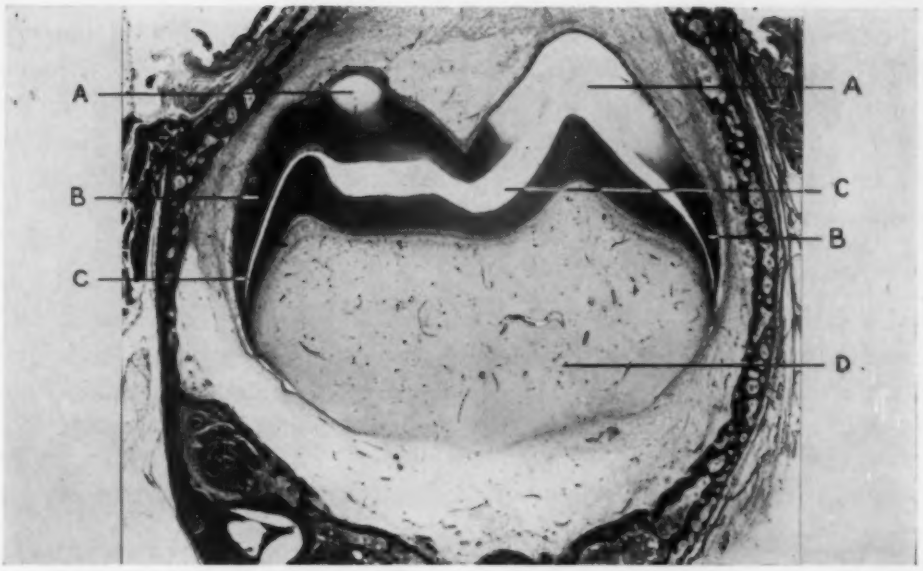


Fig. 2.—A mandibular deciduous first molar tooth germ showing the direction of calcification from the highest point of the summits of the cusps toward the dentino-enamel junction. We note at *A* (right), a complete maturation or calcification of the buccal cusp, and continuing to radiate in a direction transverse to the incremental lines. At *A* (left), we note the beginning of calcification at the summit of the lingual cusp proceeding in a similar direction toward the dentino-enamel junction. *B*, Organic enamel matrix. *C*, Characteristic shrinkage artifact. *D*, Pulp.

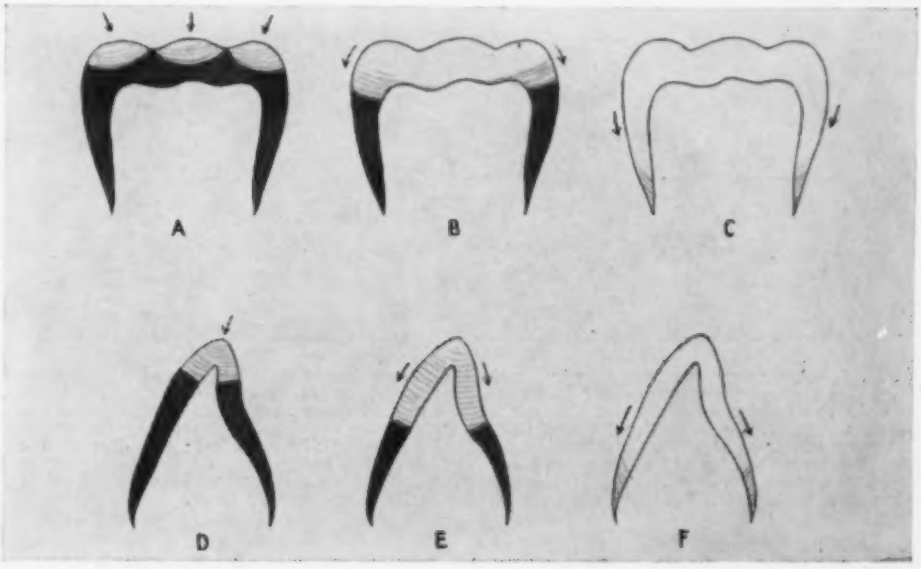


Fig. 3.—A diagrammatic representation indicating the direction of calcification following the formation of the complete organic enamel matrix. *A*, *B*, and *C* are mesiodistal views of a central incisor showing at *A*, completed calcification in the region of the mamelons of the incisal surface; the direction of radiation of such calcification indicated by the arrows. *B* shows the entire incisal surface calcified; the direction of such calcification indicated by the arrows and by the faint transverse line. Note the blunt line of separation between fully calcified enamel and the organic enamel matrix. *C* shows the peripheral surfaces fully calcified; the direction of such calcification indicated by the arrows, and by the faint transverse line in the cervical region. *D*, *E*, and *F* represent a labiolingual section diagrammatically of an incisor tooth. It is easy to follow the direction of calcification which, at *D*, involves the incisal tip. At *E*, the progress of such calcification is noted. At *F*, the peripheral surfaces of the enamel are completely calcified. The light transverse lines further help to indicate the direction of calcification.

ings indicate that two entirely different groups of deficiency lesions may occur; one, commonly called hypoplasia, is a deficiency of formation of the organic enamel matrix; the other is a failure of crystallization or calcification of the colloidal calcium contained in the organic matrix. The histogenesis of deficiency lesions of the organic matrix has been, for the first time, clearly presented in the human embryo. These findings demonstrate that the initial lesion in the enamel-forming cells (the ameloblasts) is produced in varying degrees of destruction of the varying intensity of metabolic disturbance. A mild constitutional disturbance results in a slight irritation of the ameloblasts, and a grave metabolic disturbance may result in the complete destruction of those ameloblasts which are functioning to produce enamel at that particular time. Their findings also show that an important phenomenon associated with the disturbances of the ameloblasts is an exudation of enamel secretion from the protoplasm of the cell. In mild cases of irritation, they found very slight globules of such enamel secretion exuded from the body of the protoplasm; and in more extreme cases of ir-

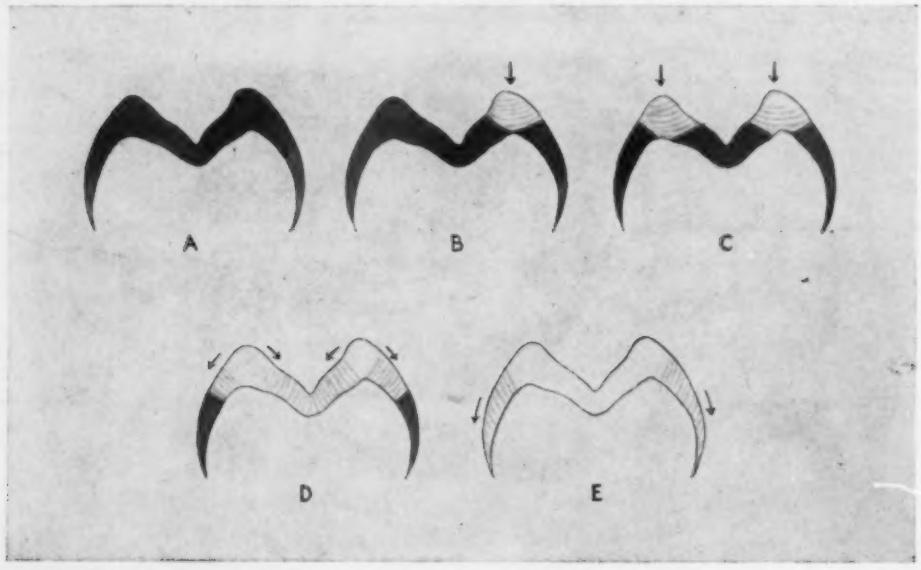


Fig. 4.—A diagrammatic series indicating the direction of calcification of a posterior tooth. *A* shows the full adult thickness of the organic enamel matrix before calcification begins. At *B*, we note the calcification of the summit of the buccal cusp and its direction is indicated by the arrows. At *C*, we note the beginning of calcification of the lingual cusp; the direction is noted by its arrows. *D* shows the entire occlusal surface fully calcified; the direction of such calcification indicated by the arrows and by the light lines radiating in the direction transverse to the incremental lines. *E*, the peripheral surfaces calcified as far as the cervical region; the direction again indicated by the arrows, and by the light transverse lines.

ritation, evidence shows the entire amount of enamel secretion lawlessly expelled from the disrupted cells, and deposited in a formless mass upon the layer of enamel matrix already formed. In such a localized area, where the cells are completely degenerated, no additional enamel matrix could form; this results in the eventual clinically appearing hypoplasias. In instances of mild irritation of short duration, the ameloblasts may recover and go on to produce normal enamel matrix, leaving behind an increased incremental line as permanent evidence of its period of irritability. This type of deficiency lesion, which is called hypoplasia, is therefore a deficiency of formation of the organic enamel matrix.

The second type of lesion, which has been mentioned earlier as a failure of crystallization of the calcium salts contained in the organic enamel matrix, results in leaving the enamel in a matrix state. Such failure of crystallization may be noted in circumscribed or localized areas, and in extreme instances, the entire enamel may present such a picture. The enamel is therefore left in a matrix state of completion and becomes more susceptible to external, mechanical, and possibly bacterial factors.

The Mechanical and Histologic Problems in the Rotation of Teeth: By Kaare Reitan, Norway, *Den Norske Tannl. Tid.* **50**: 1 (January), 1940.

The right and left second incisors of the maxilla in dogs were used to determine changes taking place in the supporting tissues of teeth during rotation. These teeth were banded and small staples were soldered on the lingual and labial sides of the bands. The canines were used as anchor teeth.

To avoid injury of the soft tissues and interference with the mandibular teeth during mastication, the mandibular incisors were ground off repeatedly. The force applied was measured by means of a special apparatus and was varied in the different cases from 9 to 250 Gm., while the time interval used for the rotation varied from three to fifty-nine days.

Histologically, there is a distinction between the tissue reactions called forth by a continuous force (steel appliances in which auxiliary springs of a thickness of 0.020 to 0.030 mm. are used) and those produced by a, mechanically speaking, intermittent force such as ligatures.

When a continuous force is applied, there is less "jiggling"; the tooth is quite firm during rotation and moves faster even if a relatively weak force is applied, due to the fact that the auxiliary springs are activated over a longer period.

The periodontal fibers, which are supposed to undergo interstitial growth when continuous force is applied, become severely stretched and arrange themselves more or less in the direction of the movement of the tooth, running diagonally from the alveolar wall to the surface of the root. The capillaries are found more or less compressed and, together with the fibroblasts, are arranged parallel to the periodontal fibers. At the same time, new osteoid tissue is formed in the tension areas in the form of tongue-like trabeculae, often followed by a compensatory resorption of the outer bone wall on the labial side. This new bone is transformed and rounded off to form a new *lamina dura* during the period of retention and layers of cellular cement are formed on the tension surface of the roots, especially in the middle and apical portions.

By applying an intermittent force, mechanically speaking, the periodontal fibers are exposed to a similar stretching, while the alveolus as a whole is considerably widened, particularly on the labial side, and the tooth is loosened.

Since the subepithelial tissues are not subject to rapid transformation, an elastic contraction of the relatively loose connective tissue may take place when the force is removed if a sufficient period of retention is not allowed. This is possibly one of the principal causes of relapse after rotation.

Considering the rather different anatomic conditions found in man and in the dog, the result of the experiments presented may be summed up as follows:

1. The form of the tooth must be taken into consideration if rotation is to be undertaken. It is possible that a tooth, the root of which in cross section more nearly approximates the arc of a circle, could be rotated within the periodontal space without causing any resorption of the root surface.

2. Due to the form of the teeth rotated in the experiments, root resorption occurred somewhat in almost every case.

3. With the appliances used in the present experiments, the teeth were more or less tipped during rotation.

4. A trabecular tongue-like arrangement of bone takes place on the tension sides of the root. These bone lamellae are transformed and smoothed out during the period of retention according to the new position of the tooth. Cellular cementum is deposited in a marked degree on the tension side of the root, while there is none or little cementum formed on the pressure side.

5. Relapse after tooth rotation is due to the lack of functional balance between new and old tissue, and—in addition to the role played by the tissue fluid and the transformation of the fibers—is influenced by the apparent elastic contraction of the subepithelial tissue.

6. In order to avoid "jiggling," sufficient stable appliances with the correct amount of anchorage should be used.

7. And, from the viewpoint of mechanics, intermittent force or a continuous force, followed by rest periods, should be applied to avoid root resorption and to allow the surrounding tissues to readjust themselves.

Management of Facial Paralysis by Physical Measures: By M. K. Newman, J. M. Berris, and S. S. Bohn, Detroit. *Archives of Physical Therapy* Chicago **21**: 257 (May), 1940.

Newman and his co-workers managed thirty cases by the use of physical therapeutic measures. The ages of the patients ranged between 6 and 79 years, average 37. With the onset of an acute refrigerans type of facial paralysis iodides in saturated solution of potassium or syrup of hydriotic acid are indicated for their general alterative action. Saline laxatives are used during the acute stage of inflammation for their possible detergent effect.

The physical measures applicable during the acute phase of the refrigerant palsies are directed toward the reduction of the inflammatory exudate and toward keeping the denervated muscles in position of physiologic rest. Decongestion at the stylomastoid foramen can be accomplished by cantharides plasters, hot moist applications, radiant heat, negative galvanism, and conventional or short wave diathermy. The frequency of application is indicated by the pain and the tolerance of the tissues.

To prevent muscles from stretching they are supported by attaching two Y bands of adhesive tape at the zygomatic bone and temporal region. Positive galvanism is especially efficacious for the relief of pain. With the subsidence of the acute symptoms within eight to ten days, testing for reaction of degeneration is instituted. When no reaction of degeneration is present, recovery can be expected in from two to eight weeks. If a sufficient amount of recovery is not obtained after six months of rigorous physical therapeutic measures, surgery is to be considered.

Active treatment now consists of heat, massage, voluntary exercise and electrical stimulation (with interrupted galvanic, sinusoidal and faradic currents). Diathermy or radiant heat is applied to the paralyzed side of the face for from fifteen to thirty minutes. Another method of promoting a circulatory response consists of the common ion transfer 0.5 per cent acetyl-beta-methylcholine hydrochloride. From 15 to 20 milliamperes of the galvanic current for twenty minutes is adequate. Negative galvanism for its heating effect can be applied for thirty minutes over the stylomastoid foramen. The strength of current should be just less than the amount required to cause a contraction of the muscles on the unaffected side. On the first day three contractions over the motor points are sufficient, and on subsequent days the number of contractions is increased but always kept within the fatigue range of the muscle. After one or two weeks the slow sine wave is substituted, from twenty to thirty contractions per minute being used.

When the faradic current can be tolerated, it should replace the sine wave current. Gentle stroking massage which follows stimulation preserves muscular tone. Exercise of the facial muscles is carried out in front of a mirror every day. The etiologic agent must always be removed before physical measures are instituted. Seventeen of the thirty cases followed exposure, six middle ear disease, one dental manipulation, one trauma associated with an automobile accident, two head colds, one mumps, one an operation on the eighth nerve for vertigo, one a post-partum infection and two mastoidectomy. In the seventeen cases of exposure paralysis, a partial reaction of degeneration or no reaction of degeneration was present after ten days. Eight of these patients received only heat, massage, and exercise, while the remainder received electrical stimulation as well.

Contribution to the Pathology and Treatment of the Intermittent Swelling of the Parotid Gland: By Stephan Rosenak, M.D., Budapest, *Monatsschrift für Ohrenheilkunde und Laryngo-Rhinologie* No. 2, 1939.

Besides chronic inflammatory changes of the glandular tissue, the intermittent swelling of the parotid gland is due to a mechanical but incomplete occlusion of the excretory duct in the majority of cases. During periods of rest the duct has an adequate capacity for salivary secretion. During increased activity, while chewing, etc., the narrowed duct does not meet the requirements of increased output of saliva. Besides organic strictures there exists a reflex "nervous" contraction of the excretory duct in certain cases. The author refers to case reported by Wolf in which a swift swelling of both submaxillary glands was noted on introduction of a Rehfuss tube; and to the case of Horváth, in which a similar sudden swelling of the submaxillary glands was observed following the placing of a cotton swab for dental treatment. He has the impression that slight inflammatory changes of the buccal mucosa may evoke a reflex contraction of the opening of Stenson's duct. In about 60 per cent of these cases the catheterization of the papilla may bring relief giving way to the accumulated and concentrated saliva behind the papilla. In 40 per cent of cases repeated dilatation of the papilla alone cannot definitely relieve the

duct tree of the abnormal pressure. The saliva consequently becomes more viscous, and the secretion of saliva becomes more and more difficult.

In order to break this vicious circle the author sharply pierced the opening of Stenson's duct by means of a 1 mm. edge-pointed knife. In four cases thus treated the viscosity of the saliva decreased, and its outflow remained continuous. The swelling of the corresponding gland decreased immediately and its size was reduced to normal within a few weeks. The wound of the mucous membrane healed within 3 to 4 days. Before performing this harmless operation one must ascertain that the narrowing of the salivary papilla is to be blamed for the stoppage of saliva and the swelling of the gland and that the latter does not occur as a consequence of inflammatory changes of the gland itself.

Summary.—Chronic intermittent swelling of the parotid gland is due mostly to a partial stenosis of the opening of Stenson's duct. This may result as a nervous reflex action following inflammatory changes of the oral mucous membrane. Repeated catheterization of the duct is essential and in 40 per cent of cases the sharp piercing of the stenosis is to be done to obtain the unhindered outflow of saliva and to avoid recurrences.

Editorials

Orthodontic Safety-First Theme

IN THE urge and quest for advancement of practice as a science and art a realization of the hazards of practice is too often overlooked. Teachers, even, in their zeal to inspire the student to give all to the job in hand, often fail to impress the student with the fact that his calling is circumscribed by grave dangers as well as compensations and satisfactions.

A man fell off a shed in a Middle Western town recently and when he awakened in the hospital, he learned that his hip was fractured and that his lower denture was missing. A thorough search failed to locate the teeth. The condition became grave, consultation was called, and treatment for diphtheria was started all to no avail. Specialists were called in desperation to save a life, and an x-ray promptly disclosed that in the trachea reposed the missing teeth. The teeth were removed with the Jackson instrument, and the patient promptly recovered, after having been practically asphyxiated.

A patient of an orthodontist is reported to have caught the free end of a premolar clasp of a Hawley retainer over the distal edge of the soft palate, and in an attempt to remove the clasp, the soft palate was perforated. Had it not been for a resourceful father with his automobile pliers, another asphyxiation might have resulted from the careless (sans safety first) construction of an orthodontic appliance.

With the widening scope of orthodontic practice, more and more cases are reported of bands, free ends of separating ligatures, free end finger springs, etc., being lodged in the pharynx, frequently resulting in panic of children and parents. Orthodontic appliances, particularly anterior bands and spurs, are of perfect size and architecture to be easily breathed into the trachea and upper lung, and when once lodged there, they are difficult to remove. One experience will quickly impress the orthodontist that by far the most important part of his practice is to leave no stone unturned to prevent his patient from inhaling minor fragments of metal while under his care. Teachers should lay more stress on the hazards of orthodontic practice on this score, for these are not generally discussed as much as they should be in order to create the caution necessary to develop a *safety-first* program of practice.

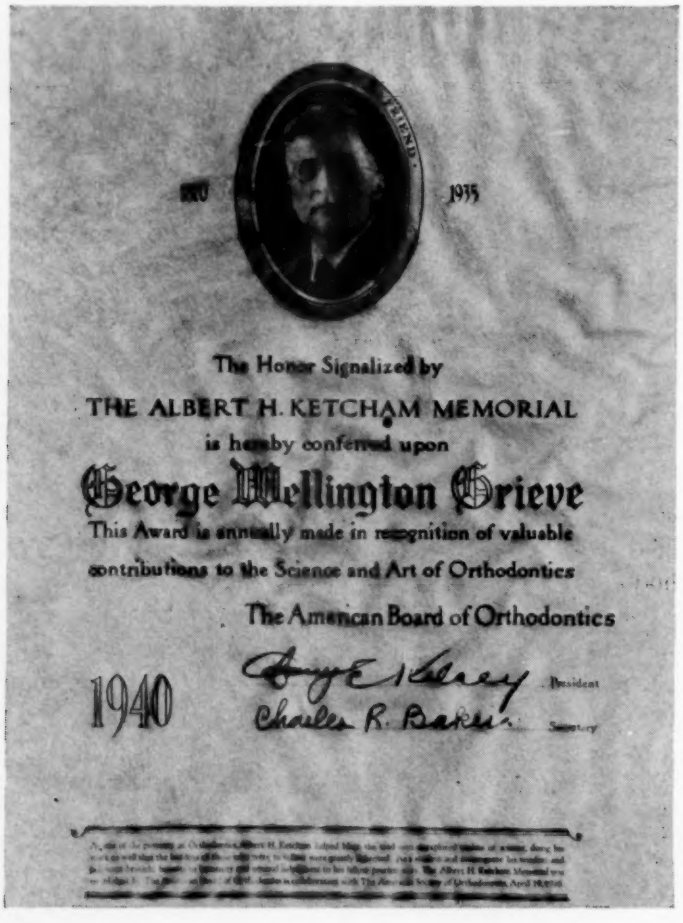
One experience with asphyxiation is a nightmare to any practitioner, and his patient as well. In no better place than this can the old adage, "An ounce of prevention is worth a pound of cure," be applied. Caution, care, and a condition of safety-first mindedness developed in the orthodontist are just as important as skill in practice—perhaps more important. Now is the time to make orthodontists more safety conscious before there are more asphyxiations as a result of delicate appliances becoming dislodged during both the sleeping and waking hours. Orthodontics is due for a safety-first campaign, among its own workers as an efficiency measure in practice.

H. C. P.

In reporting the Ketcham Award as conferred upon Dr. George Grieve in the August issue of the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY, the remarks of response by Dr. Grieve were not included because the transcript of this material was not received early enough to go to press. Accordingly, the remarks pertaining to the acceptance follow.

REMARKS OF DR. GEORGE W. GRIEVE IN ACCEPTING THE KETCHAM AWARD

Mr. Chairman, Members of the Board of Orthodontics, and Members of the American Association of Orthodontists, I thank you sincerely for the signal honor which you have conferred upon me.



The Ketcham Award presented to Dr. George W. Grieve. (Courtesy of the *Journal of the Canadian Dental Association.*)

When I received notice from Doctor Baker, Secretary of the Board, that I was to be this year's recipient of the Ketcham Award, I was probably more surprised than I had ever been before in my life, as I know many men whom I believe to be more worthy. I appreciate the honor of being associated with those men who have already received this Award. As we get on in life, an honor such as this is much more appreciated than almost anything that could

happen to us in the way of recognition of honest effort. I mean you will appreciate it more when you are older than when you are young.

As a young boy I had some difficulties to overcome, in an effort to make my way in the world. As quoted by Dr. Kelsey from my letter to him, some mention of this "might prove a stimulation to young men to realize that *ambition* is a major factor in success." Endowed with reasonably good health, one will accomplish what he wants to do if he has the ambition to work for it.

I naturally feel very keenly the honor bestowed upon me here tonight. I admired Dr. Ketcham very much. Mrs. Grieve and I had the pleasure of spending a few days with Dr. and Mrs. Ketcham at their summer home at Lake Edith, in the mountains some distance from Denver, the year the A. A. O. met at Estes Park. It was a great shock to us when Albert passed on, because we were very fond of the Ketchams.

Placed "on the spot," as I am this evening, you all know how difficult it is to collect one's thoughts and express them satisfactorily. I might say that I have had some views which did not meet with general approval, but, as Dr. Kelsey has said, I was open to conviction. It is a great thing to have something to go after, and for many years I have been looking for the cause of our many failures, and with success.

I would like to quote just a few lines from a poem which I love very much. The first knowledge I had of this poem was two lines in a paper by Dr. B. E. Lischer. I was so impressed that I wrote Dr. Lischer to find out something about it, and he very kindly sent me a copy of the poem, entitled "Heroism." It is from *In This Our World,* by Charlotte Perkins Stetson.

> It takes great strength to bring your life up square
> With your accepted thought, and hold it there;
> Resisting the inertia that drags back
> From new attempts to the old habit's track.
> It is so easy to drift back, to sink;
> So hard to live abreast of what you think:
>
> * * * * * * * * *
>
> Our sun's wide glare, our heaven's shining blue,
> We owe to fog and dust they fumble through;
> And our rich wisdom that we treasure so
> Shines from the thousand things that we don't know.
> But to think new—it takes a courage grim
> As led Columbus over the world's rim.
> To think, it cost some courage, and to go—
> Try it. It taxes every power you know.

I thank you.

News and Notes

Dr. F. B. Noyes Retires as Dean

Dr. Frederick B. Noyes, a leader in the specialty of orthodontics, who has just retired as dean of the University of Illinois College of Dentistry, was honored at a banquet Sept. 14 at the Lake Shore Athletic Club, in Chicago.

Dr. Noyes was born in Chicago in 1872. The city or its suburbs has always been his home. He has been a dentist for half a century, and associated with dental education for all but the first six years of that time. For seventeen years he was a prominent member of Northwestern University's dental school faculty; however, during the last twenty-seven years he has been at the University of Illinois College of Dentistry.

Dr. Noyes received his first dental training in the Chicago College of Dental Surgery, attended L. P. Haskell's postgraduate school of dentistry, studied at the Johns Hopkins University, then enrolled in medicine and dentistry at Northwestern University. After receiving the doctor of dental surgery degree there, he continued studies in the Angle School of Orthodontia in St. Louis, Mo.

FREDERICK B. NOYES

Dr. Noyes is author of a dental textbook which has printed five editions, and he is coauthor of another. He has received honorary degrees from Northwestern University and Temple University, and has been awarded the Callahan and Jarvey medals for distinguished accomplishment.

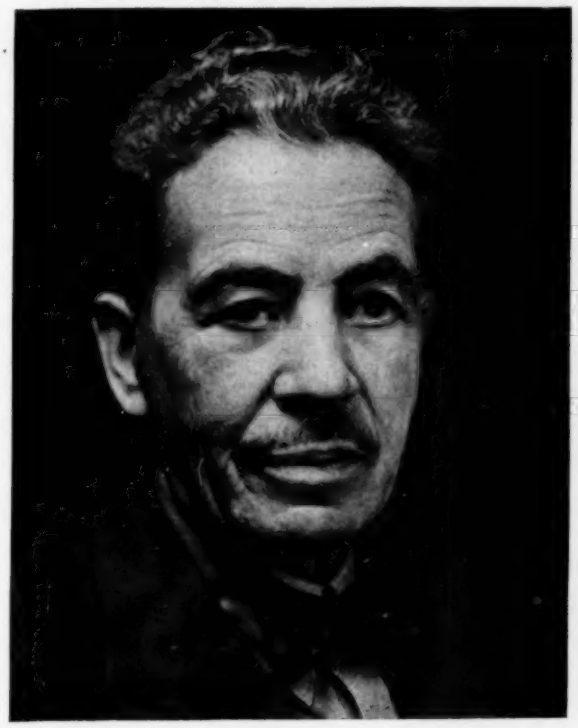
He is prominent in the Chicago Dental Society, Illinois State Dental Society, American Dental Association, Institute of Medicine of Chicago, Chicago Association of Orthodontists, Angle Society of Orthodontia, Odontographic Society of Chicago, Odontological Society of

Chicago, International Association for Dental Research, is a fellow in the American Medical Association, and an honorary member of the American Association of Orthodontists and the American College of Dentists.

Julius Aderer

Julius Aderer, president of the dental supply firm of Julius Aderer of New York, and Mrs. Aderer were killed July 22, when an amphibian plane in which they were taking their first airplane ride crashed in landing in Vineyard Haven Harbor, Mass.

Mrs. Aderer's niece, Ruth Weckman, 6 years old, daughter of Frederick L. G. Weckman, treasurer of the Aderer firm, was also a passenger in the plane. She was knocked unconscious in the crash, but was extricated from the wreckage by Charles G. Frederick, pilot of the plane. Mr. Frederick, who suffered severe head and body bruises, pulled Mr. and Mrs. Aderer, as well as the girl, from the plane which sank within three minutes after the crash and kept them afloat until a speed boat arrived from the Vineyard Haven Yacht Club.



JULIUS ADERER

Mr. Aderer was one of the first to introduce platinum gold alloys for use in orthodontics. At his death he was 73 years old, and he will be long remembered as one of the pioneers in orthodontic manufacturing and supply business.

Great Lakes Association of Orthodontists

The fourteenth annual meeting of the Great Lakes Association of Orthodontists will be held at the Royal York Hotel, Toronto, Ont., Oct. 28 and 29, 1940.

Mid-Continent Dental Congress

The Mid-Continent Dental Congress will be held in St. Louis, Mo., at the Hotel Jefferson, Oct. 27 to 30, 1940.

OFFICERS OF ORTHODONTIC SOCIETIES*

American Association of Orthodontists

President, Henry U. Barber, Jr. - - - - - 5 East Fifty-Seventh St., New York, N. Y.
Secretary-Treasurer, Max E. Ernst - - - 1250 Lowry Medical Arts Bldg., St. Paul, Minn.
Public Relations Bureau Director, Dwight Anderson
- - - - - - - - - - - - - - - - - - 292 Madison Ave., New York, N. Y.

Central Association of Orthodontists

President, Harold J. Noyes - - - - - - - - 30 N. Michigan Ave., St. Paul, Minn.
Secretary-Treasurer, L. B. Higley - - - - - - - - 705 Summit Ave., Iowa City, Iowa

Great Lakes Association of Orthodontists

President, Harvey G. Bean - - - - - - - - - - - 170 St. George St., Toronto, Can.
Secretary-Treasurer, Richard E. Barnes - - - - - - - Republic Bldg., Cleveland, Ohio

Harvard Society of Orthodontists

President, I. D. Davis - - - - - - - - - - - - - - - 419 Boylston St., Boston, Mass.
Secretary-Treasurer, Edward I. Silver - - - - - - - - 80 Boylston St., Boston, Mass.

New York Society of Orthodontists

President, Glenn F. Young - - - - - - - - - - - 745 Fifth Ave., New York, N. Y.
Secretary-Treasurer, William C. Keller - - - - 40 E. Forty-Ninth St., New York, N. Y.

Pacific Coast Society of Orthodontists

President, Will G. Sheffer - - - - - - - - - - - Medico Dental Bldg., San Jose, Calif.
Secretary-Treasurer, Earl F. Lussier - - - - - - 450 Sutter St., San Francisco, Calif.

Rocky Mountain Society of Orthodontists

President, Leonard T. Walsh - - - - - - - - - - - - Thatcher Bldg., Pueblo, Colo.
Secretary-Treasurer, George Siersma - - - - - - - 1232 Republic Bldg., Denver, Colo.

Southern Society of Orthodontists

President, A. C. Broussard - - - - - - - 1116 Maison-Blanche Bldg., New Orleans, La.
Secretary-Treasurer, T. C. Sparks - - - - - - - 1508 Washington St., Columbia, S. C.

Southwestern Society of Orthodontists

President, G. C. Turner - - - - - - - - - - - - 406 Myrick Bldg., Lubbock, Texas
Secretary-Treasurer, R. E. Olson - - - - - - - Union Nat'l Bank Bldg., Wichita, Kan.

Washington-Baltimore Society of Orthodontists

President, George M. Anderson - - - - - - - - - - 831 Park Ave., Baltimore, Md.
Secretary-Treasurer, Stephen C. Hopkins - - - - - 1726 Eye St., Washington, D. C.

American Board of Orthodontics

President, Harry E. Kelsey - - - - - - - - - - - - 833 Park Ave., Baltimore, Md.
Vice-President, Frederic T. Murlless, Jr. - - - - - 43 Farmington Ave., Hartford, Conn.
Secretary, Charles R. Baker - - - - - - - - - - - 636 Church St., Evanston, Ill.
Treasurer, Bernard G. DeVries - - - - - - - Medical Arts Bldg., Minneapolis, Minn.
William E. Flesher - - - - - 806 Medical Arts Bldg., Oklahoma City, Okla.
Frederic T. Murlless, Jr. - - - - - - 43 Farmington Ave., Hartford, Conn.
Oliver W. White - - - - - - - - 213 David Whitney Bldg., Detroit, Mich.
James D. McCoy - - - - - - - - 3839 Wilshire Blvd., Los Angeles, Calif.

Foreign Societies†

British Society for the Study of Orthodontics

President, S. A. Riddett
Secretary, R. Cutler
Treasurer, Harold Chapman

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

†The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 8022 Forsythe, St. Louis, Mo., U. S. A.